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Table of Contents

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ORIGINAL ARTICLES— PAGE.

"The Abuse of the Drainage Tube in the Treatment of Peritonitis, with an Analysis of 244 Cases of Diffuse Peritonitis Treated without Drainage", by ALFRED J. TRINCA, M.D., F.R.C.S., F.R.A.C.S. 465

"Early Diagnosis and Treatment of Uterine Cancer", by H. H. SCHLINK, M.B., Ch.M., F.R.A.C.S., and CLEMENT L. CHAPMAN, D.S.O., V.D., M.B., Ch.M., F.R.C.S. (Eng.), F.R.C.S. (Edin.) 476

"Dental Health Education in New South Wales", by E. BRANGWIN GREEN 482

REPORTS OF CASES—

"Closure of a Defect in the Hard Palate by a Pedicled Tube Graft", by G. H. BURNELL, M.D., B.S., F.R.C.S., F.R.A.C.S. 484

REVIEWS—

Minor Ailments 484
The Cellular Action of Drugs 485
The Classification and Diagnosis of Heart Disease 485
Materia Medica and Therapeutics 486
Dermatology and Its History 486

LEADING ARTICLES—

Preventive Dentistry 487

CURRENT COMMENT—

Cardiovascular Syphilis 488
Changes in the Liver in Catarrhal Jaundice 489

ABSTRACTS FROM CURRENT MEDICAL LITERATURE— PAGE.

Medicine 490

BRITISH MEDICAL ASSOCIATION NEWS—

Scientific 492
Nominations and Elections 494

MEDICAL SOCIETIES—

The Melbourne Pædiatric Society 495

OBITUARY—

William David Kerr Macgillivray 496

CORRESPONDENCE—

Pathological Examination of Tumours 497
The Problem of Gastric Carcinoma 498
Diagnostic Swabbings in Diphtheria 499
The Use of Iodine in Thyreotoxicosis 499
Influenza 499

DIARY FOR THE MONTH 500

MEDICAL APPOINTMENTS 500

MEDICAL APPOINTMENTS VACANT, ETC. 500

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 500

EDITORIAL NOTICES 500

THE ABUSE OF THE DRAINAGE TUBE IN THE TREATMENT OF PERITONITIS, WITH AN ANALYSIS OF 244 CASES OF DIFFUSE PERITONITIS TREATED WITHOUT DRAINAGE.[1]

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THE treatment of peritonitis has been a matter of controversy ever since active interference by the surgeon became the established practice. General agreement has been reached concerning the necessity for immediate operation with a view to eradicating the primary focus of infection in cases of peritonitis, but the question of drainage is still a matter of contention, although the advocates of non-drainage are gradually gaining adherents.

It is the object of this paper to try by argument and a study of the results obtained in the treatment of 244 cases of diffuse peritonitis by non-drainage to prove that the use of the drainage tube in the treatment of the majority of cases of peritonitis is not only futile and unnecessary, but may be productive of great harm. Divergent views are also held in connexion with other procedures that should be adopted at the time of operation, particularly the treatment of the exudate, and there exist also differences of opinion with regard to the post-operative treatment. It is claimed that the principles of treatment carried out at the time of operation and the post-operative management in this series of cases are rational and explain the low mortality and the small incidence of serious complications.

The study of statistical records of appendicitis, which is the primary cause of the great majority of

[1] Read at a meeting of the Victorian Branch of the British Medical Association on July 5, 1933.

cases of peritonitis, shows that there has been no appreciable reduction in the mortality from this disease during the last twenty years. The mortality among patients operated upon within the first twenty-four hours remains almost negligible, but rises suddenly to a high figure in those operated upon on the second and third days, and still higher a few days later. Whilst a certain mortality must remain inevitable when advanced peritonitis results from delay in treatment, it is my contention that the mortality in appendicitis can be considerably reduced by lessening the number of deaths that at present occur in the earlier, curable stages of peritonitis.

In other words, there is no excuse for the high mortality among patients operated upon on the second and third days of the disease. This high figure is a serious reflection on the present surgical management of peritonitis.

The idea of non-drainage in peritonitis is not new. Mickulicz,[1] in 1885, successfully treated a case of perforation of the small intestine by closure of the peritoneal cavity without drainage. Others, following in his footsteps, met with disaster because of the prevailing ignorance of aseptic technique, and the method fell into disrepute, to be revived again in 1911.

During the last twenty years there have been many advocates of non-drainage, and in spite of hostile criticism, the trend, particularly in the last ten years, shows an increase in the number who recommend this procedure where possible.

In spite of this, there are still many surgeons in this country (I believe the majority, and they include many of our younger men) who are obsessed with the imaginary danger and virulent nature of inflammatory exudates in the abdominal cavity, and resort to the use of the drainage tube in all cases of peritonitis. Others go a step further, and after removing the primary focus insert a drainage tube even where no exudate is present, because they feel that the primary focus, usually a gangrenous appendix, must have infected the peritoneal cavity. Their guiding principles are apparently: "When in doubt, drain"; or that when pus is present, it is highly infective and must be let out.

The Peritoneal Cavity.

The peritoneal cavity is only a potential space, owing to the nature and distribution of its contents. The distensible hollow viscera contain gas at atmospheric pressure, and therefore lie in intimate contact with one another and the solid viscera and parietes. There is no space between them, except that occupied by the omentum and the small quantity of natural peritoneal secretion. Any congestive or inflammatory exudate can find a place only by displacing the gas in the hollow viscera or by distending the abdominal wall. In many inflammatory exudates, owing to peritoneal spoiling, there occurs in a few hours a deposit of adhesive lymph and loculation of exudate in the pelvis, abdominal fossæ, and between coils of small intestine, the process being helped by the omentum when large enough. The peritoneal cavity, however, still remains only a potential cavity.

In the light of modern knowledge of the physiology and pathology of the peritoneum, many of the old ideas which were thought to be established and which formed the basis of the earlier lines of treatment, must be abandoned. Contrary to what was originally taught, absorption takes place readily from all parts of the peritoneal surface.[2] Some other explanation, therefore, must be given to account for the beneficial results arising from the adoption of the Fowler position in the treatment of peritonitis. Recovery is not due to a lessened capacity of the pelvic peritoneum to absorb noxious substances. The peritoneal lining of the small intestine absorbs toxic substances that act on the ganglion cells in the plexus of Auerbach, and so produce ileus, which is the cause of death rather than septicæmia in the majority of cases. The higher the ileus, the more serious the results, and, by taking advantage of gravity, by allowing the exudate to gravitate as far as possible into the pelvis, as in the Fowler position, a few coils of ileum, rather than many coils of jejunum, bear the brunt of the attack. It is well recognized that the effects of the obstruction of the small intestine vary in severity directly with the proximity to the beginning of the small intestine. This may be an explanation of the benefit resulting from the adoption of the Fowler position. Absorption from the peritoneum is delayed in cases of inflammation, and when an exudate is very fibrinous and abundant there is practically no absorption.[3]

The degree of purulence is generally an index of the resisting power of the patient rather than of the virulence of the infection. A notable example is in the case of streptococcal peritonitis, where the exudate remains thin and there is little or no fibrin formation, death occurring very rapidly in most cases from a rapidly spreading subperitoneal cellulitis. This forms a separate type of peritonitis and will not be considered in this paper.

The omentum has abundant absorptive powers.[2] Experiments with the intraperitoneal injection of virulent bacteria show that at the time of death of an animal the exudate may be sterile and yet numerous bacteria and abundant cultures may be obtained from the omentum. The significance of this will be discussed later in dealing with treatment. Bacteriological investigation of the exudate in the majority of cases of peritonitis in man gives exactly similar results, the inflammatory exudate being either sterile or yielding only a feeble growth on attempts at cultivation on suitable media. Some years ago, in a series of cases of peritonitis following appendicitis, I made smears and cultures of the exudate and was surprised to find that the smears contained apparently intact normal staining leucocytes with no evidence of toxic spoiling, that organisms were often absent or difficult to find, and that when they were present no indication of their numbers could be obtained from cultures made from

the same exudate. There was either no growth or else considerably fewer colonies appeared than the number seen in smears would lead one to expect. This is evidence that the exudate is really a protective leucocytic and serological barrier. Its removal is not only unnecessary, but harmful, except in so far as it obscures the surgeon's field of vision or is contaminated with fragments of necrotic material or fæcoliths, as in cases of advanced gangrene or perforation of the appendix, or foreign material from a perforated viscus. The toxic spoiling of the endothelial lining of the visceral and parietal peritoneum will still remain for some time after removal of the exudate, and a further quantity of serous fluid will be poured out and its amount probably increased as a result of the added trauma produced by the surgeon in his ill-advised attempts to remove as much as possible of the original exudate. It will result in a serious dehydration, which the patient can ill withstand.

The tolerance of the peritoneum to infection is not appreciated by the surgeon who attempts to drain the peritoneal cavity in all cases of peritonitis. The peritoneum can stand a sledge hammer blow of infection from the majority of pathogenic organisms, provided the blow is not repeated too often or the attack is not too prolonged. Once a focus of infection has been adequately dealt with, such as by the removal of a gangrenous appendix, or the hole in a perforated viscus has been closed, the protective exudate poured out in abundance can deal with the infection. It is necessary only to stop the attack or, if this is impossible, to direct its effects away from the general peritoneal cavity.

To sum up: Ignoring of the elementary physical fact that the peritoneal cavity containing hollow viscera is only a potential space, lack of knowledge of the true pathology of peritonitis and of the tolerance of the peritoneum to infection, and an exaggerated idea of the virulence of the organisms present in most inflammatory peritoneal exudates have been responsible for the introduction and continued use of the drainage tube in the treatment of peritonitis.

The Futility of Attempted Drainage.

A very little contemplation will show that it is impossible adequately to drain the peritoneal cavity. Attempts at drainage are futile and may be productive of great harm. When a drainage tube is inserted into a peritoneal exudate, the open end, if not placed against the posterior abdominal wall, and the side openings, if any, are immediately closed by the application of an adjoining coil of intestine or a piece of omentum. The only fluid which escapes from the tube whilst it is in position is that which entered it during the process of insertion, or that which makes its way at the side of the tube. On removing the tube one or several days later, there is a gush of imprisoned fluid produced as an inflammatory reaction around a foreign body which would not be tolerated by a healthy peritoneum, much less by one in a state of inflammation and toxic spoiling. The surgeon has increased the degree and amount of peritonitis rather than lessened it.

The Exudate.

It has already been pointed out that the more abundant and the more fibrinous the exudate, the less the absorption. Frank pus in the peritoneal cavity is very often "laudable pus" and, with certain reservations, should be left undisturbed. Its presence should not produce panic, with its attendant rapid swabbing out or removal by other means, and the insertion of one or more large drainage tubes. If the bacterial nature of a peritoneal exudate is of such a virulence that the resistance of the patient cannot overcome it, removal of some of the exudate will not save the patient. It is quality, not quantity, that counts. In other words, a very few virulent organisms, such as a virulent streptococcus which is unresisted and which tends to grow in the peritoneal cavity, will upon entry inevitably kill the patient. The most strenuous endeavours on the part of the surgeon to remove all of the infective material will fail, and the small quantity left will continue the fatal process in spite of the drainage tube.

The Harmful Effects of the Drainage Tube.

The harm done by drainage tubes introduced into the abdominal cavity will naturally increase with the length of time the tube is left in.

We can summarize as follows the possible evil effects:

1. *Aggravation of the Existing Peritonitis.*—Aggravation of the existing peritonitis has already been mentioned in the discussion, where it is stated that the drainage tube is a foreign body. Fortunately, in the great majority of cases the accumulated exudate from this cause remains localized, but it may remain unabsorbed because of the added damage to the injured peritoneum and produce a residual abscess.

2. *Fæcal Fistula.* The incidence of fæcal fistula will vary with the length of time the tube is left in, and on the presence or absence of side openings in the tube. The contact of the end or side openings of the tube with a coil of intestine may produce pressure necrosis. It has already been mentioned that the openings in a tube are immediately closed by the application of the nearest coil of intestine or omentum. Adhesions form in a few hours, delicate at first, but firm after the second day. It can easily be understood how removal of the tube can split or tear away a piece of the bowel wall with the formation of a fæcal fistula.

3. *Damage to Omentum.*—In the same way it is quite possible to lacerate a portion of adherent omentum. I have seen one case of fatal omental hæmorrhage result from the removal of a tube which had been left in five days.

4. *Damage to Abdominal Wall.*—The tissues of the parietes will not tolerate a foreign body any more than any other tissues, and the immediate

effects are the production of the phenomenon of inflammation, with congestion, cellular infiltration and exudation of such a degree that pressure necrosis occurs around the tube, the necrotic tissue forming a pabulum for infecting organisms present in the exudate and the skin. A slough is the result, with the production of a hole larger than the drainage tube through all layers of the abdominal wall, including the peritoneum. In addition, infection around the tube is pent up and further increases the tension and spread of superficial inflammation or cellulitis.

5. *Post-Operative Hernia.*—Closure of the peritoneum after drainage is by inelastic scar tissue or the wall of the bowel supported by scar tissue—very poor representatives of the original peritoneum and four layers of muscle.

6. *Adhesions and Intestinal Obstruction.*—Among the causes of obstruction of the small intestine post-operative adhesions rank high, and a large proportion of these result from drainage of the abdominal cavity in cases of peritonitis.

7. *Introduction of Organisms from the Air.*—Occasionally an intractable sinus is left after drainage of the peritoneum. A change in character of the exudate and the findings on culture suggest, in such cases, that there has been a superadded infection from the outside. I have seen two cases of *Bacillus pyocyaneus* infection of an appendiceal sinus.

8. *Ileus.*—I have no doubt, from a consideration of the cases that I have treated without drainage, irrespective of the cause or duration of the peritonitis before operation, that the drainage tube is a potent cause of ileus. I have for comparison cases in which drainage was considered necessary, and ileus in many of these has been a worrying factor; the relief of ileus has been observed after the removal of the tube. The tube acts in two ways: (i) as an irritant to the coils of small intestine with which it is in contact, and (ii) by the formation of adhesions and kinks which produce intestinal obstruction.

9. *Delayed and Painful Convalescence Following Some or All of the Above Evils.*—The net result will be prolonged stay in hospital, a long period of invalidism and economic loss. I have in mind a man who, three months ago, was seized with an attack of acute appendicitis.

Six hours after the onset he was operated upon and, as "he was on the verge of peritonitis" (to quote the surgeon's actual words), a drainage tube was placed in the pelvis and one in the flank, and both were left in for five or six days. At present he is still an invalid with two fæcal fistulæ and two painful abdominal wounds, and has lost his employment.

Circumstances in Which Drainage is Considered Necessary.

The circumstances in which drainage is considered necessary may be summarized as follows:

1. *Where the Infective Focus has not been Eradicated.*—In appendicitis it is occasionally impossible, owing to the patient's condition or technical difficulties to remove the appendix completely or in part; or it may be impossible, owing to a patient's condition, to effect closure of a perforated viscus. In cases such as these a tube should be introduced, precautions being taken to see that it accurately reaches the focus of infection and is left in long enough for a protective barrier to form. On removing the tube on the second day after operation, a track is left, along which infective material from the focus can travel to the outside.

2. *Free Fæcoliths.*—Occasionally a perforated appendix is found, and it is suspected that a fæcolith has escaped through the perforation. A search should be made for this infective foreign body and, if not found, it is safer to leave a tube in the pelvis, where the fæcolith is most likely to gravitate. The same applies when a friable appendix breaks up during removal and portions of gangrenous *débris* escape, usually to the bottom of the pelvis.

3. *Raw Areas.*—Where the appendix is buried in adhesions and after removal a raw, oozing area bereft of peritoneum is left. This particularly applies when the retroperitoneal tissues have been exposed. Here my usual practice is to insert a gauze drain through a stab wound in the flank lateral to McBurney's incision. The lower end is packed against the oozing surface and the gauze is removed in from eighteen to twenty-four hours after operation. Where there is no hæmorrhage a piece of rolled-up rubber dam will suffice.

(In appendiceal abscess we have a walled-off, localized collection of pus with a persistent cavity isolated from the general peritoneal cavity, and it is not subject to the same physical laws as the normal peritoneal cavity. Here a drainage tube will not be futile.)

The Surgical Management of Peritonitis.

It is not claimed that the low mortality rate in this series is due to any special technique or particular operative skill, but solely to what is, in my opinion, the rational treatment of peritonitis.

Procedure at the Time of Operation.

The lightest possible anæsthesia should be employed, gas and oxygen in the more serious cases. It is essential to perform the operation as rapidly and delicately as possible, avoiding the undue use of retractors and packs, unnecessary exposure, and rough handling or exposure of the intestine. Whenever possible, the appendix should be removed *in situ*. In even the early stages of peritonitis there is endothelial spoiling of the parietal and especially of the visceral peritoneum, and the employment of packs to swab out peritoneal exudate and rough packing off of coils of intestine severely damage this endothelium and are a potent cause of persistent post-operative adhesions and ileus.

Treatment of the Exudate.

If the primary focus of infection has been completely removed, there is no necessity to remove any except that which may obscure the field of operation. The fluid is protective in nature.

There are some exceptions however. Sometimes an appendix is very friable and fragments of gangrenous *débris* or thick, blood-stained or foul pus are found mixed with the fluid, thus constituting themselves part of the primary focus; or a hole in a perforated appendix suggests that a fæcolith has escaped. In the first example the fluid mixed with gangrenous *débris* should be gently removed by a suction pump, the nozzle of which rests among the surgeon's fingers to prevent contact and injury to coils of intestine. In the second instance a search should be made for the suspected fæcolith, exudate being removed to facilitate the search.

The position can be summed up by saying that if the exudate is homogeneous, none need be removed, but if mixed with gangrenous *débris* or visceral contents or what is considered infective material from the primary focus, all should be removed.

A criticism that has been levelled against the leaving of the peritoneal exudate *in situ* is that the exudate is liable to become loculated and form residual or pelvic abscesses. It is my opinion that this complication will not occur in the absence of drainage if the infective focus is totally eradicated. This is borne out by the series of cases tabulated. There was only one case of pelvic abscess, which was drained by a posterior colpotomy operation, and a fæcolith was discharged with the first gush of pus.

Treatment of the Omentum.

Sodden, stiffened or gangrenous omentum is found at times wrapped around or adherent to a gangrenous appendix. The omentum has the capacity to absorb pathogenic organisms from the peritoneal cavity, and complete removal of the primary infective focus in cases of gangrenous appendicitis should therefore include removal of any doubtful omentum. In cases of appendiceal abscess which have been treated by simple drainage, occasionally a piece of gangrenous omentum is discharged as a slough. It will be appreciated then that neglect in removal of involved omentum may result in persistence and spread of the peritonitis or the formation of a residual abscess.

Post-Operative Complications.

Ileus.—Paralytic ileus in this series was not a common complication, in spite of the duration of the peritonitis in many of the cases. A certain degree of distension is present, of course, in most cases of peritonitis, due to toxæmia, but I am convinced that ileus can be made to increase and to become paralytic and often fatal by rough handling at the time of operation, by the introduction of a foreign body in the shape of a drainage tube and by unscientific or meddlesome after-treatment.

Wound Infection.

The natural resistance of the tissues of the abdominal wall is not so great as that of the peritoneum to organisms introduced from the hollow viscera, and it is inevitable that in a certain proportion of cases wound infection will occur. The use of a drainage tube will inevitably cause wound infection, because, in addition to the infection from the peritoneal exudate, there is the added element of lowered resistance and tissue strangulation or necrosis produced by the drainage tube.

Precautions Against Wound Infection.

Certain precautions can be taken to minimize the incidence of wound infection.

1. *The Use of Skin Packs Before Opening the Peritoneum.*—The use of skin packs before opening the peritoneum, of course, only prevents gross contamination by gangrenous *débris* or fæcoliths, the fluid exudate quickly soaking through the pack. If care be taken to wrap a gangrenous appendix, when delivered through the wound, in a small pack or piece of gauze, the wound will run still less risk of contamination.

2. *Avoidance of Unnecessary Dissection and Opening Up of Tissue Planes in Opening the Abdomen.*—It is unwise to make a dissecting room display in making McBurney's incision. There is no need, after cutting through the skin and layers of subcutaneous tissues, to make by blunt dissection an exposure of a large area of external oblique aponeurosis. This only opens up a large space for the accumulation of blood or of pus in the cases of subsequent infection. I take the greatest care never to expose any but a thin band of aponeurosis in the line of the incision, and the same applies after incising the aponeurosis. It is unwise to clean up the fibres of the internal oblique and to separate them widely from the overlying external oblique; nor is it necessary after exposing the peritoneum to make a dead space by separating from it in all directions the overlying transversalis aponeurosis. Dead spaces should be avoided at all stages of opening the abdominal wall.

3. *Careful Hæmostasis.*—A common cause of wound infection is an infected hæmatoma. Even if the connective tissues have a natural resistance, the blood forms a nidus for the growth of the colon bacillus and other organisms.

Points on After-Treatment.

Immediately after operation the patient is given one or more rectal injections of saline solution or saline solution by the drip method, the amount of solution depending on the severity of the infection and the tolerance of the patient. If the rectal administration of saline solution, either intermittent or continuous, causes any distress, the treatment is stopped. The Fowler position is adopted, but not kept up during the whole course of the convalescence. In a mild case this is needed for a few days only; in a case with ileus it is needed until the ileus has abated.

The treatment of paralytic ileus can be summed up by saying: "Do as little as possible." The administration of gas anti-serum, introduced by B. W. Williams,[3] has been a most valuable aid in the treatment of this serious complication, and since its introduction in 1926 it has been used in

some of the cases that showed ileus, by the daily intramuscular or intravenous injection of 10,000 concentrated units until the bowels open. Before this special remedy was tried, however, ileus was not considered as irremediable, and we were dependent on what is considered a still more important aspect of the management of ileus, and that is the withholding of all forms of fluid and solids by the mouth and the withholding of purgatives and involuntary muscle stimulants, such as pituitrin and eserine. Frequent sips of water on the day of or day following operation and the use of a soda water siphon should be particularly forbidden. These frequent sips are usually accompanied by air swallowing in the case of an exhausted patient and produce vomiting. Further, the administration of any form of nourishment by the mouth increases or sets up intestinal paresis. An inflamed part needs rest, not stimulation.

The Use of Morphine.

I do not believe in the continuous routine use of morphine on the lines advocated in recent years. It is given with the idea of resting an inflamed part. The inflamed bowel is already at rest; in fact, it is often paralysed. Why give unnecessary drugs? Morphine is a very valuable drug in the treatment of peritonitis, but why give it in excess? Sufficient morphine to produce at least six to eight hours' sleep in the twenty-four has been found quite sufficient in this series. It is used to give mental rest, not intestinal. It is claimed by the advocates of the continuous use of morphine that it relieves pain by stopping painful peristalsis. Pain after operation has not been a striking feature in this series. Little or no evidence of painful peristalsis was noted, and it is my belief that painful peristalsis is produced in response to the irritation of one or more drainage tubes inserted among coils of intestine.

Purgatives and Intestinal Stimulants.

Purgatives, speaking generally, are either intestinal irritants or stimulants of intestinal peristalsis. Their use in the after-treatment of peritonitis, especially when ileus is present, cannot be too strongly condemned. I feel quite sure that many lives are lost by their administration. Nature is clamouring for rest for the inflamed bowels. Whipping the tired horse produces no response. Purgatives will often produce an ileus not previously present, and will certainly increase any ileus already present.

In ileus the patient should be left severely alone as far as the bowels are concerned for days if necessary, and it will be found that suddenly the bowels are opened naturally in most cases and several fluid motions may be passed in the twenty-four hours. In mild cases an enema is administered on the third day and alternate days after that. Often, however, natural bowel actions occur without any interference.

One of the beneficial results of gas gangrene antiserum treatment is the restoration of tone to the paralysed bowel, with the resumption of peristalsis and natural bowel action.

Intestinal stimulants, such as pituitrin and eserine, on the same lines of argument, have no place in the treatment of peritonitis. If ileus be present, they are ineffective; if not, they are quite unnecessary.

In my opinion, the administration of purgatives and intestinal stimulants in the treatment of peritonitis is a therapeutic crime.

Commentary on Statistical Table.

Table I represents a consecutive series of cases of diffuse peritonitis from various causes taken over the period October, 1926, to May, 1933. The majority represent patients admitted to the Alfred Hospital, but a few patients operated on in private practice during the same period have been also included. Peritonitis has been classified into pelvic and general, pelvic meaning that the exudate did not reach above the anterior superior iliac spine, and general that the exudate reached above this level. As Sampson Handley[4] points out, so-called general peritonitis is very rarely universal, reaching to the dome of the diaphragm. Mild cases of peritonitis in its early stages, in which there was only a small collection of serous fluid at the bottom of the pelvis, were not included. In all cases operated upon there was sufficient fluid present to escape when the peritoneum was opened.

Cases of pelvic peritonitis arising from tubal infections, such as gonorrhœa, which had been operated upon as the result of a mistaken diagnosis of appendicitis, were not included, because these cases tend to subside without operation.

In the majority of cases the fluid in the peritoneal cavity was not removed.

Drainage was not employed, except in four cases of gangrene of the gall-bladder, where a small tube was introduced into the gall-bladder fossa after cholecystectomy to drain away the possible escape of bile.

In every case where the appendix was the primary cause of the peritonitis, the appendix was gangrenous wholly or in part, or it was perforated. In a few cases the gangrene was confined to the mucosa.

Appendiceal abscess was included only when it burst into the general peritoneal cavity and produced a spreading peritonitis. In all of these cases the wall of the appendiceal abscess was formed entirely of omentum and the abscess was excised *in toto*.

No cases of pneumococcal or fatal streptococcal peritonitis were encountered during this period.

Most of the patients were adults, there being only eleven children in the series under the age of thirteen years.

With regard to the duration of the peritonitis, it was impossible to determine exactly when the peritonitis supervened in the cases in which the appendix was the primary cause. The figures given under the heading "duration of disease" refer to

TABLE I.

244 Cases of Peritonitis from Various Causes Treated by Operation Without Drainage.

No. of Case.	Initials.	Age.	Sex.	Duration.	Primary Cause.	Type of Peritonitis and Exudate.	Operation.	Post-Operative Complications.	Days in Hospital.	Result.	Date.
1	L.G.	36	M.	6 hours.	Perf. duod. ulcer.	Genl. perit., sero-purulent.	Simple closure.	Slight wound infection.	18	Cured.	3.10.25
2	F.E.	15	M.	17 hours.	Gang. append.	Pelvic perit., sero-purulent.	Appendicectomy.	—	14	Cured.	9.10.25
3	E.J.	54	M.	3 days.	Gang. append.	Genl. perit., purulent.	Appendicectomy.	Wound infection.	30	Cured.	10.10.25
4	W.S.	30	M.	10 hours.	Gang. append.	Pelvic perit., sero-purulent.	Appendicectomy.	—	11	Cured.	17.10.25
5	R.M.	23	M.	30 hours.	Gang. append.	Pelvic perit., sero-purulent.	Appendicectomy.	—	18	Cured.	9.11.25
6	W.C.	27	M.	?	Gang. append.	Pelvic perit., sero-purulent.	Appendicectomy.	—	10	Cured.	23.11.25
7	F.D.	27	M.	*21 days.*	Rupt. append. abscess.	Genl. perit., thick pus.	Excision of abscess.	Slight wound infection.	23	Cured.	30.11.25
8	R.S.	15	M.	36 hours.	Gang. append.	Pelvic perit., sero-purulent.	Appendicectomy.	—	16	Cured.	8.12.25
9	G.H.	20	F.	3 days.	Gang. append.	Pelvic perit., purulent.	Appendicectomy.	—	10	Cured.	14.12.25
10	M.S.	45	F.	2 days.	Perfd. diverticulum.	Pelvic perit., purulent.	Excision of diverticulum.	—	20	Cured.	2.1.26
11	A.J.	47	M.	3 days.	Gang. append.	Genl. perit., purulent.	Appendicectomy.	Wound infection.	22	Cured.	4.1.26
12	R.D.	54	M.	16 hours.	Gang. append.	Pelvic perit., sero-purulent.	Appendicectomy.	—	13	Cured.	4.1.26
13	G.McC.	36	M.	2 days.	Rupt. append. abscess.	Pelvic perit., containing faecoliths.	Excision of abscess.	—	13	Cured.	15.2.26
14	N.D.	16	M.	36 hours.	Gang. append.	Pelvic perit., sero-purulent.	Appendicectomy.	—	9	Cured.	1.3.26
15	J.E.	25	M.	18 hours.	Gang. append.	Pelvic perit., sero-purulent.	Appendicectomy.	—	14	Cured.	5.5.26
16	C.B.	23	M.	24 hours.	Gang. append.	Genl. perit., sero-purulent.	Appendicectomy.	—	10	Cured.	31.5.26
17	J.T.	28	M.	12 hours.	Gang. append.	Pelvic perit., sero-purulent.	Appendicectomy.	—	10	Cured.	11.6.26
18	W.B.	52	M.	12 hours.	Perfd. ileum by chop bone.	Genl. perit., sero-purulent.	Removal of bone, closure of ileum.	Wound infection.	23	Cured.	14.6.26
19	E.F.	25	F.	10 hours.	Gang. append.	Pelvic perit., purulent.	Appendicectomy.	—	12	Cured.	21.6.26
20	H.S.	50	M.	3 days.	Gang. append.	Genl. perit., purulent.	Appendicectomy.	—	16	Cured.	28.6.26
21	E.M.	35	F.	72 hours.	Gang. gall-bladder.	Genl. perit., purulent.	Cholecystectomy, drainage of gall-bladder fossa.	Wound infection.	21	Cured.	7.7.26
22	F.E.	29	M.	50 hours.	Gang. append.	Pelvic perit., purulent.	Appendicectomy.	—	11	Cured.	12.7.26
23	J.B.	24	M.	12 hours.	Gang. append.	Pelvic perit., purulent.	Appendicectomy.	—	12	Cured.	29.7.26
24	W.V.	38	M.	4 days.	Gang. append.	Pelvic perit., purulent.	Appendicectomy.	—	11	Cured.	19.8.26
25	C.J.	28	M.	48 hours.	Gang. append.	Pelvic perit., purulent.	Appendicectomy.	—	11	Cured.	19.8.26
26	J.J.	56	M.	36 hours.	Gang. append.	Pelvic perit., purulent.	Appendicectomy.	—	10	Cured.	24.8.26
27	M.T.	29	F.	15 hours.	Gang. append.	Pelvic perit., purulent.	Appendicectomy.	—	11	Cured.	5.9.26
28	A.C.	67	M.	12 hours.	Gang. append.	Pelvic perit., sero-purulent.	Appendicectomy.	Wound infection.	19	Cured.	20.9.26
29	H.H.	36	M.	2 days.	Gang. append.	Pelvic perit., sero-purulent.	Appendicectomy.	—	10	Cured.	28.9.26
30	A.W.	55	M.	8 hours.	Perf. duod. ulcer.	Genl. perit., sero-purulent.	Simple closure.	—	27	Cured.	30.9.26
31	E.P.	22	M.	17 hours.	Gang. append.	Pelvic perit.	Appendicectomy.	Pyelitis, pneumonia.	49	Cured.	30.9.26
32	N.H.	31	M.	16 hours.	Gang. append.	Genl. perit., purulent.	Appendicectomy.	—	23	Cured.	8.10.26
33	E.F.	50	M.	6 days.	Gang. append.	Genl., purulent.	Appendicectomy.	—	17	Cured.	18.10.26
34	N.E.	25	F.	36 hours.	Gang. append.	Genl., sero-pus.	Appendicectomy.	Wound infection (pregnant)	37	Cured.	18.10.26
35	W.B.	22	M.	19 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	13	Cured.	25.10.26
36	K.C.	25	F.	36 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	22	Cured.	30.10.26
37	E.H.	21	M.	16 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	14	Cured.	4.11.26
38	R.B.	41	M.	2 hours.	Perf. duod. ulcer.	Genl., serous.	Simple closure.	—	20	Cured.	6.11.26
39	E.B.	17	F.	?	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	10	Cured.	7.11.26
40	S.O.	17	M.	12 hours.	Gang. append.	Genl., sero-pus.	Appendicectomy.	—	13	Cured.	22.11.26
41	M.C.	15	M.	36 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	10	Cured.	2.12.26
42	E.S.	16	F.	20 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	11	Cured.	3.12.26
43	E.T.	33	M.	3 days.	Gang. append.	Genl., purulent.	Appendicectomy.	—	14	Cured.	20.12.26
44	S.L.	44	M.	14 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	17	Cured.	20.12.26
45	H.W.	38	M.	?	Ruptd. append. abscess.	Genl., purulent.	Excision abscess.	—	15	Cured.	10.1.27
46	H.S.	13	M.	?	Gang. append.	Genl., sero-pus.	Appendicectomy.	—	15	Cured.	29.1.27
47	J.D.	25	M.	48 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	20	Cured.	19.2.27
48	G.K.	18	M.	3 days.	Gang. append.	Pelvic, purulent.	Appendicectomy.	Wound infection.	24	Cured.	19.2.27
49	D.L.	18	F.	14 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	14	Cured.	5.2.27
50	C.N.	27	F.	36 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	13	Cured.	28.2.27
51	C.P.	19	M.	14 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	13	Cured.	2.3.27
52	R.C.	40	M.	13 hours.	Gang. append.	Genl., sero-pus.	Appendicectomy.	Wound infection.	30	Cured.	8.3.27
53	P.G.	56	F.	10 hours.	Gang. gall-bladder.	Genl., purulent.	Cholecystectomy.	Wound infection.	30	Cured.	12.3.27
54	C.B.	45	M.	20 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	14	Cured.	21.3.27
55	E.R.	24	F.	3 days.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	12	Cured.	26.3.27
56	C.O'B.	18	F.	?	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	28.3.27
57	C.J.	26	M.	36 hours.	Gang. append.	Pelvic, foul pus.	Appendicectomy.	Wound infection.	30	Cured.	4.4.27
58	S.T.	25	M.	2 days	Gang. append.	Genl., purulent.	Appendicectomy.	Wound infection.	20	Cured.	19.4.27
59	R.T.	20	M.	24 hours.	Gang. append.	Genl., sero-pus.	Appendicectomy.	—	12	Cured.	2.5.27
60	H.L.	19	M.	3 days.	Gang. append.	Genl., purulent.	Appendicectomy.	—	12	Cured.	23.5.27
61	D.V.	27	F.	12 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	23	Cured.	23.5.27
62	G.R.	49	M.	12 hours.	Perf. duod. ulcer.	Genl., sero-pus.	Simple closure.	Pneumonia 5 days post-operative.	13	*Death.*	30.5.27
63	J.C.	55	M.	12 hours.	Gang. append.	Genl., sero-pus.	Appendicectomy.	Wound infection.	20	Cured.	30.5.27
64	P.N.	33	F.	24 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	35	Cured.	17.6.27
65	M.McQ.	23	F.	24 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	14	Cured.	20.6.27
66	W.McK.	44	M.	?	Gang. append.	Genl., purulent.	Appendicectomy.	—	20	Cured.	20.6.27
67	W.G.	23	M.	48 hours.	Rupt. append. abscess.	Genl., purulent.	Excision abscess.	Wound infection.	29	Cured.	27.6.27

Perfd.—Perforated. duod.—duodenal. Genl.—General. perit.—peritonitis. Gang.—Gangrenous. append.—appendix. Ruptd.—Ruptured.

TABLE I.—*Continued.*

344 Cases of Peritonitis from Various Causes Treated by Operation Without Drainage.—Continued.

No. of Case.	Initials.	Age.	Sex.	Duration.	Primary Cause.	Type of Peritonitis and Exudate.	Operation.	Post-Operative Complications.	Days in Hospital.	Result.	Date.
68	J.B.	12	M.	?	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	Wound infection.	20	Cured.	4.7.27
69	R.B.	14	M.	3 days.	Gang. append.	Genl., purulent.	Appendicectomy.	Wound infection.	36	Cured.	16.7.27
70	D.D.	19	F.	24 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	13	Cured.	17.7.27
71	O.P.	32	M.	3 days.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	18	Cured.	18.7.27
72	O.B.	27	M.	24 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	11	Cured.	18.7.27
73	McC.	38	F.	?	Gang. append.	Genl., sero-pus.	Appendicectomy.	—	10	Cured.	5.8.27
74	Y.B.	23	F.	18 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	14	Cured.	22.7.27
75	H.W.	20	M.	12 hours.	Gang. gall-bladder.	Genl., purulent.	Cholecystectomy.	—	17	Cured.	1.8.27
76	N.A.	12	F.	3 days.	Ruptd. append. abscess.	Genl., purulent.	Excision abscess.	Wound infection.	32	Cured.	7.8.27
77	T.S.	24	M.	32 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	13	Cured.	13.8.27
78	E.B.	25	M.	15 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	12	Cured.	18.8.27
79	R.W.	28	M.	9 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	14	Cured.	13.9.27
80	J.C.	12	F.	6 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	12	Cured.	3.11.27
81	F.W.	23	M.	24 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	Wound infection.	28	Cured.	6.11.27
82	G.F.	35	M.	15 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	Slight wound infection.	17	Cured.	16.11.27
83	W.D.	10	M.	12 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	12	Cured.	21.11.27
84	C.P.	41	M.	24 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	13	Cured.	28.11.27
85	R.B.	24	M.	12 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	10	Cured.	12.12.27
86	J.M.	15	M.	36 hours.	Gang. append.	Genl., sero-pus.	Appendicectomy.	—	10	Cured.	20.12.27
87	S.H.	30	M.	3 days.	Gang. append.	Genl., thick pus.	Appendicectomy.	Wound abscess.	12	Cured.	29.12.27
88	C.F.	33	M.	24 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	9	Cured.	2.1.28
89	N.W.	17	F.	24 hours.	Gang. append.	Genl., sero-pus.	Appendicectomy.	—	9	Cured.	2.1.28
90	G.H.	40	M.	36 hours.	Ruptd. append. abscess.	Genl., sero-pus.	Excision abscess.	—	6	*Death.*	30.1.28
91	E.T.	23	F.	36 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	Wound infection.	20	Cured.	6.2.28
92	G.D.	37	M.	12 hours.	Gang. append.	Genl., sero-pus.	Appendicectomy.	Femoral thrombosis.	47	Cured.	13.2.28
93	M.M.	15	F.	14 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	20.2.28
94	O.P.	?	F.	?	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	21.2.28
95	E.G.	23	F.	20 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	27.2.28
96	J.K.	68	M.	2 days.	Gang. append.	Genl., purulent.	Appendicectomy.	Wound infection.	32	Cured.	5.3.28
97	B.T.	40	M.	8 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	14	Cured.	8.3.28
98	T.B.	36	M.	10 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	11	Cured.	12.3.28
99	J.B.	21	F.	17 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	8	Cured.	19.3.28
100	N.R.	19	M.	12 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	12	Cured.	23.4.28
101	R.P.	43	M.	11 hours.	Gang. append.	Genl., sero-pus.	Appendicectomy.	Wound infection.	21	Cured.	23.4.28
102	W.B.	19	M.	6 hours.	Perfd. duod. ulcer.	Genl., sero-pus.	Simple closure.	—	17	Cured.	13.5.28
103	G.N.	57	M.	12 hours.	Perfd. duod. ulcer.	Genl., purulent.	Simple closure.	—	25	Cured.	20.5.28
104	D.F.	19	F.	48 hours.	Ruptd. append. abscess.	Pelvic, purulent.	Excision abscess.	—	12	Cured.	25.5.28
105	J.S.	52	M.	5 hours.	Perfd. duod. ulcer.	Genl., serous.	Simple closure.	—	12	Cured.	29.5.28
106	G.H.	35	F.	12 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	16	Cured.	4.6.28
107	A.S.	13	M.	8 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	13	Cured.	17.6.28
108	F.S.	38	M.	30 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	13	Cured.	18.6.28
109	H.H.	42	M.	?	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	23.7.28
110	A.H.	36	M.	16 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	21	Cured.	25.7.28
111	E.R.	21	F.	18 hours.	Gang. append.	Genl., sero-pus.	Appendicectomy.	—	14	Cured.	23.9.28
112	P.K.	13	F.	48 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	15	Cured.	5.10.28
113	J.R.	22	M.	18 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	Wound infection.	24	Cured.	8.10.28
114	V.N.	38	M.	56 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	Wound infection.	32	Cured.	8.11.28
115	F.H.	62	M.	?	Gang. append.	Genl., sero-pus.	Appendicectomy.	—	15	Cured.	13.11.28
116	J.McK.	33	M.	3 days.	Gang. append.	Genl., thick pus.	Appendicectomy.	—	4	*Death.*	2.12.28
117	W.L.	36	M.	12 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	12	Cured.	3.12.28
118	H.O.	42	M.	16 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	20.12.28
119	McL.	30	M.	10 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	12	Cured.	25.1.29
120	D.P.	23	M.	3 days.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	10	Cured.	12.2.29
121	D.J.	14	F.	48 hours.	Gang. append.	Genl., sero-pus.	Appendicectomy, post colpotomy.	Pelvic abscess.	46	Cured.	22.2.29
122	D.G.	14	F.	24 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	12	Cured.	24.2.29
123	P.C.	22	M.	15 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	25.3.29
124	W.B.	9	M.	8 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	10.4.29
125	E.W.	28	M.	19 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	21	Cured.	19.4.29
126	A.W.	41	F.	20 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	Wound infection.	33	Cured.	15.5.29
127	J.C.	36	M.	16 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	18	Cured.	13.5.29
128	C.J.	27	M.	15 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	8	Cured.	13.5.29
129	R.B.	35	M.	10 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	Wound infection.	17	Cured.	10.6.29
130	T.P.	26	M.	24 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	11	Cured.	20.7.29
131	T.W.	79	M.	4 days.	Gang. append.	Pelvic, thick pus.	Appendicectomy.	Wound infection.	22	Cured.	29.7.29
132	E.G.	42	F.	36 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	Wound infection.	16	Cured.	14.7.29
133	H.H.	14	M.	2 days.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	11	Cured.	4.8.29
134	S.B.	16	M.	12 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	11	Cured.	4.8.29
135	H.F.	52	M.	6 hours.	Perfd. gastric ulcer.	Genl., sero-pus.	Simple closure.	Burst wound, re-suture wound.	27	Cured.	11.8.29
136	B.D.	23	F.	6 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	5.8.29
137	C.P.	37	F.	18 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	Wound infection.	17	Cured.	9.8.29

Perfd.—Perforated. duod.—duodenal. Genl.—General. perit.—peritonitis. Gang.—Gangrenous. append.—appendix. Ruptd.—Ruptured.

TABLE I.—*Continued.*

244 Cases of Peritonitis from Various Causes Treated by Operation Without Drainage.—Continued.

No. of Case.	Initials.	Age.	Sex.	Duration.	Primary Cause.	Type of Peritonitis and Exudate.	Operation.	Post-Operative Complications.	Days in Hospital.	Result.	Date.
138	T.L.	40	M.	8 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	14	Cured.	8.9.29
139	I.R.	42	M.	11 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	14	Cured.	8.10.29
140	R.W.	43	M.	20 hours.	Perfd. duod. ulcer.	Genl., purulent.	Simple closure.	—	4	*Death.*	27.1.30
141	T.H.	20	M.	18 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	8	Cured.	27.1.30
142	J.S.	17	M.	30 hours.	Ruptd. append. abscess.	Genl., purulent.	Excision abscess.	Wound abscess.	14	Cured.	22.2.30
143	P.G.	35	M.	4 hours.	Perfd. duod. ulcer.	Genl., serous.	Simple closure.	Wound infection.	20	Cured.	27.2.30
144	R.F.	36	M.	11 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	14	Cured.	18.2.30
145	E.S.	46	F.	24 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	10	Cured.	9.3.30
146	E.B.	36	F.	3 days.	Ruptd. append. abscess.	Genl., purulent.	Excision abscess.	—	14	Cured.	16.3.30
147	F.B.	42	F.	24 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	11	Cured.	21.3.30
148	O.C.	12	F.	14 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	14	Cured.	22.3.30
149	R.G.	14	M.	30 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	8	Cured.	7.4.30
150	G.B.	35	M.	18 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	16	Cured.	22.4.30
151	McD.	35	M.	11 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	14	Cured.	3.6.30
152	McK.	23	F.	8 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	12	Cured.	13.8.30
153	J.H.	43	M.	2 days.	Gang. append.	Genl., purulent.	Appendicectomy.	Wound infection.	10	Cured.	18.8.30
154	O.C.	21	F.	5 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	8	Cured.	18.8.30
155	A.C.	20	F.	10 hours.	Gang. append.	Genl., sero-pus fæcoliths.	Appendicectomy.	—	10	Cured.	26.7.30
156	F.D.	29	M.	36 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	11	Cured.	1.9.30
157	N.B.	19	M.	2 hours.	Perfd. rectum.	Pelvic, sero-pus fæces.	2 ft. wire removed; rectum closed.	Hæmorrhage from wound.	15	Cured.	5.9.30
158	M.M.	46	M.	12 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	Wound infection.	14	Cured.	9.9.30
159	E.M.	29	M.	12 hours.	Gang. append.	Pelvic, sero-pus fæcoliths.	Appendicectomy.	Wound infection.	38	Cured.	6.10.30
160	McK.	38	F.	18 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	Wound infection.	23	Cured.	15.10.30
161	Mc.	18	F.	13 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	14	Cured.	16.10.30
162	J.N.	36	M.	?	Perfd. duod. ulcer.	Genl., sero-pus.	Simple closure.	—	20	Cured.	27.10.30
163	A.C.	62	M.	3 hours.	Perfd. duod. ulcer.	Genl., serous.	Simple closure.	Wound infection.	19	Cured.	2.11.30
164	J.W.	18	M.	14 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	11	Cured.	2.11.30
165	O.M.	32	F.	36 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	2.11.30
166	G.C.	13	M.	36 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	Wound infection	15	Cured.	10.11.30
167	H.B.	27	M.	24 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	10.11.30
168	M.S.	17	F.	19 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	9	Cured.	8.12.30
169	C.B.	30	M.	15 hours.	Gang. append.	Pelvic, purulent fæcoliths.	Appendicectomy.	Wound infection.	15	Cured.	15.12.30
170	A.C.	76	M.	4 days.	Gang. append.	Genl., thick pus.	Appendicectomy.	—	6	*Death.*	14.1.31
171	J.L.	27	M.	18 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	11	Cured.	20.2.31
172	W.D.	13	M.	48 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	Wound infection.	11	Cured.	20.2.31
173	B.S.	14	F.	30 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	7	Cured.	16.3.31
174	D.H.	18	F.	14 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	6	Cured.	16.3.31
175	W.S.	42	M.	6 hours.	Perfd. gastric ulcer.	Genl., sero-pus.	Simple closure.	—	9	Cured.	21.3.31
176	H.M.	65	M.	?	Ruptd. append. abscess.	Genl., sero-pus.	Excision abscess.	—	15	Cured.	21.3.31
177	E.M.	15	M.	56 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	Wound infection.	20	Cured.	22.5.31
178	R.P.	21	M.	18 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	21	Cured.	4.6.31
179	M.J.	29	F.	16 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	14	Cured.	15.5.31
180	A.N.	55	F.	32 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	Wound infection.	28	Cured.	9.6.31
181	A.B.	10	M.	8 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	9.7.31
182	E.P.	43	F.	17 hours.	Gang. gall-bladder.	Genl., purulent blood-stained.	Cholecystectomy.	Slight wound infection.	23	Cured.	27.7.31
183	C.H.	20	M.	36 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	5	Cured.	27.7.31
184	T.L.	45	M.	11 hours.	Gang. append.	Genl., sero-pus.	Appendicectomy.	—	16	Cured.	30.7.31
185	J.B.	50	M.	30 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	8	Cured.	24.8.31
186	J.P.	41	M.	5 hours.	Perfd. gastric ulcer.	Genl., sero-pus.	Simple closure.	Bronchopneumonic empyema.	42	Cured.	24.8.31
187	H.K.	39	M.	9 hours.	Perfd. duod. ulcer.	Genl., sero-pus.	Simple closure.	—	24	Cured.	9.9.31
188	B.M.	19	F.	2 days.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	7	Cured.	7.9.31
189	M.M.	8	M.	13 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	14	Cured.	27.9.31
190	L.L.	21	M.	18 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	Wound infection.	23	Cured.	2.10.31
191	P.P.	51	M.	30 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	Wound infection.	27	Cured.	25.1.32
192	McG.	14	M.	20 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	14	Cured.	6.2.32
193	L.Y.	14	F.	*10 days.*	Gang. append.	Genl., thick pus.	Appendicectomy.	—	7	Cured.	8.2.32
194	E.C.	46	M.	3 days.	Gang. append.	Genl., purulent.	Appendicectomy.	Slight wound infection.	20	Cured.	19.2.32
195	R.T.	17	F.	2 days.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	8	Cured.	20.2.32
196	C.B.	22	M.	24 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	8	Cured.	20.2.32
197	O.McK.	18	F.	12 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	7	Cured.	7.3.32
198	G.P.	11	F.	4 days.	Gang. append.	Pelvic, purulent.	Appendicectomy.	Wound abscess.	24	Cured.	7.3.32
199	J.S.	36	F.	30 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	10	Cured.	21.3.32
200	W.M.	18	F.	8 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	12	Cured.	23.4.32
201	N.P.	18	F.	24 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	Wound infection.	17	Cured.	30.5.32
202	S.C.	40	M.	3 days.	Gang. append.	Genl., purulent.	Appendicectomy.	Wound infection.	24	Cured.	25.7.32
203	A.D.	19	F.	3 days.	Gang. append.	Genl., purulent.	Appendicectomy.	—	8	Cured.	8.8.32
204	M.D.	73	M.	?	Perfd. carcinoma of the colon.	Genl., pus and air.	Laparotomy.	—	2 hours.	*Death.*	8.8.32
205	W.P.	50	M.	4 hours.	Perfd. duod. ulcer.	Genl., serous.	Simple closure.	—	21	Cured.	15.8.32
206	J.F.	45	M.	8 hours.	Perfd. gastric ulcer.	Genl., purulent.	Simple closure.	Wound infection.	16	Cured.	16.9.32

Perfd.—Perforated. duod.—duodenal. Genl.—General. perit.—peritonitis. Gang.—Gangrenous. append.—appendix. Ruptd.—Ruptured.

TABLE I.—*Continued.*

244 Cases of Peritonitis from Various Causes Treated by Operation Without Drainage.—Continued.

No. of Case.	Initials.	Age.	Sex.	Duration.	Primary Cause.	Type of Peritonitis and Exudate.	Operation.	Post-Operative Complications.	Days in Hospital.	Result.	Date.
207	L.C.	19	M.	15 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	14	Cured.	19.9.33
208	S.S.	39	M.	36 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	7	Cured.	26.9.33
209	V.W.	50	M.	6 hours.	Perfd. duod. ulcer.	Genl., sero-pus.	Simple closure.	Wound infection.	25	Cured.	1.10.33
210	D.C.	51	M.	36 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	8	Cured.	1.10.33
211	F.S.	26	M.	?	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	8	Cured.	17.10.33
212	A.P.	13	F.	*14 days.*	Gang. append.	Genl., thick pus.	Appendicectomy.	Wound infection.	21	Cured.	17.10.33
213	H.F.	33	M.	52 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	Wound abscess.	22	Cured.	3.10.33
214	J.C.	29	M.	15 hours.	Gang. append.	Pelvic, purulent faeolith.	Appendicectomy.	—	9	Cured.	18.10.33
215	V.J.	43	M.	5 hours.	Perfd. duod. ulcer.	Genl., sero-pus.	Simple closure.	—	18	Cured.	8.10.33
216	P.N.	50	M.	22 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	8	Cured.	19.10.33
217	A.H.	66	M.	5 hours.	Perfd. gastric ulcer.	Genl., sero-pus.	Simple closure.	—	18	Cured.	26.10.33
218	F.K.	?	F.	48 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	Wound infection.	11	Cured.	16.11.33
219	E.H.	55	F.	24 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	10	Cured.	14.11.33
220	E.M.	?	F.	5 days.	Gang. append.	Genl., purulent.	Appendicectomy.	—	14	Cured.	18.11.33
221	L.B.	18	F.	24 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	5	Cured.	6.12.33
222	R.D.	13	M.	16 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	Stitch abscess.	16	Cured.	22.12.33
223	P.S.	51	F.	24 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	14	Cured.	21.11.33
224	R.J.	23	F.	13 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	14	Cured.	28.11.33
225	E.V.	52	F.	10 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	21	Cured.	6.12.33
226	M.G.	56	M.	16 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	16	Cured.	21.1.33
227	M.M.	16	M.	10 hours.	Gang. append.	Genl., sero-pus.	Appendicectomy.	—	12	Cured.	4.4.33
228	H.P.B.	8	M.	8 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	4.4.33
229	H.G.	57	F.	16 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	Wound infection.	21	Cured.	4.5.33
230	R.W.	60	M.	36 hours.	Gang. append.	Genl., purulent.	Appendicectomy.	—	13	Cured.	6.2.33
231	D.D.	38	F.	10 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	Pyelitis.	11	Cured.	6.2.33
232	R.G.	18	M.	16 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	5	Cured.	20.2.33
233	O.N.	30	M.	48 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	Wound infection.	30	Cured.	27.2.33
234	—	45	M.	36 hours.	Perfd. carcinoma of the colon.	Genl., purulent.	Closure perfn., colotomy, resection *et cetera*.	—	63	Cured.	6.3.33
235	P.B.	7	F.	36 hours.	Gang. append.	Genl., sero-pus.	Appendicectomy.	—	13	Cured.	27.3.33
236	F.E.	48	M.	19 hours.	Rupld. append. abscess.	Genl., purulent.	Excision abscess.	—	8	Cured.	10.4.33
237	E.F.	17	F.	32 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	8	Cured.	6.5.33
238	N.V.	24	F.	10 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	25	Cured.	12.5.33
239	J.H.	21	M.	16 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	Wound infection.	14	Cured.	4.11.33
240	A.L.	59	M.	12 hours.	Rupld. empyema gall bladder.	Genl., purulent, loose calculi.	Cholecystectomy.	—	21	Cured.	18.11.33
241	A.H.	26	M.	8 hours.	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	20.2.47
242	A.A.	38	F.	?	Gang. append.	Pelvic, sero-pus.	Appendicectomy.	—	10	Cured.	23.7.26
243	E.H.	35	M.	36 hours.	Gang. append.	Pelvic, purulent.	Appendicectomy.	—	12	Cured.	4.12.26
244	—	—	—	6 hours.	Perfd. gastric ulcer.	Genl., sero-pus.	Simple closure.	—	11	Cured.	14.7.26

Perfd.—Perforated. duod.—duodenal. Genl.—General. perit.—peritonitis. Gang.—Gangrenous. append.—appendix. Rupld.—Ruptured.

the period that elapsed between the onset of symptoms and the time of operation.

The longest duration stated was twenty-one days. In this case an appendiceal abscess of insidious onset had burst into the general peritoneal cavity a few days before operation. The patient was too ill for elicitation of an accurate history.

In the great majority of cases the clinical symptoms and signs of peritonitis were present at the time of operation.

Mortality.

It is claimed that the mortality rate in this series is as nearly as possible absolute. The mortality statistics of peritonitis arising from appendicitis usually given are not absolute; they state only the immediate mortality, and there will be a definite future mortality from intestinal obstruction following adhesions after the use of the drainage tube which cannot be assessed immediately after operation.

Complications.

Ileus was not common in the series and was considered serious only when it was paralytic and associated with persistent vomiting. The ileus described by some as a *sine qua non* of advanced peritonitis is, in my opinion, in a large number of cases the result of bad surgery or unscientific after-treatment.

Wound Infection.

The incidence of wound infection in this series is high. Many of the examples, however, were very mild, but in some, particularly those arising in infected hæmatomata, there was a definite increase in the length of stay in hospital. Wound infection was considered to have occurred in every case in which healing did not occur by first intention.

General.

In Case 204 (Table IV) the patient was moribund on admission and should not have been operated upon. Local anæsthesia was employed.

In Case 140 the patient walked into the casualty room of the Alfred Hospital two hours after a duodenal ulcer had perforated and refused operation. Eighteen hours later he was brought to hospital almost moribund and was operated on.

In Case 170 the patient was the oldest but one in the series.

TABLE II.

244 patients with average age 30·6 years.
(11 children under 13 years.)

Pelvic peritonitis: 124 cases.	General peritonitis: 120 cases.	Total: 244 cases.

Duration of Disease.

	Number of Cases.	Deaths.	Percentage.
Up to 24 hours	162	3	1·8
25 to 48 hours	33	1	3·0
49 to 72 hours	22	–	0
73 to 96 hours	19	1	5·2
More than 4 days ..	8	1	12·5
Total	244	6	2·46

Complications.

	Number of Cases.	Percentage.
Wound infection	52	21·3
Residual abscess	1	
Pneumonia	3	
Pyelitis	2	3·27
Femoral thrombosis	1	
Burst wound	1	
Total	60	24·5

TABLE III.

Classification of Causes of Peritonitis.

Primary Cause.	Number of Cases.	Percentage.	Number of Deaths.	Percentage.
Gangrenous appendicitis ..	202	87·35	2	1·4
Ruptured appendiceal abscess	11		1	
	213		3	
Perforated gastric and duodenal ulcers	21	8·60	2	9·95
Gangrene of the gall-bladder	4	2·0	—	—
Ruptured empyema of the gall-bladder	1		—	
Perforated viscera .. .. (ileum 1, rectum 1)	2	0·82	—	—
Perforated diverticulum ..	1	0·41	—	—
Perforated carcinoma of the colon	2	0·82	1	50
Total	244	100·00	6	2·46
Average duration of peritonitis before operation ..	23 hours.	Average stay in hospital ..		11·3 days.

TABLE IV.

Mortality Rate.

Number of Cases: 244.	Number of Deaths: 6.	Percentage: 2·46.

Mortality Analysis.

Case Number.	Initials.	Age.	Condition at Operation.	Duration.	Cause of Death.	Day of Death.	Date of Admission.
62	G.R.	49	Perforated duodenal ulcer.	12 hours.	Pneumonia 5th day after operation.	13th	30.5.27
90	G.H.	40	Gangrenous appendix, with abscess adhesion of ileum and intestinal obstruction.	36 hours.	Ileus and toxæmia.	6th	30.1.28
204	M.	78	Perforated carcinoma of the colon.	?	Toxæmia.	2 hours.	8.8.32
116	J.McK.	33	Gangrenous appendix, with general peritonitis.	3 days.	Ileus and toxæmia.	4th	2.12.28
140	R.W.	43	Perforated duodenal ulcer.	20 hours.	Toxæmia.	4th	27.1.30
170	A.C.	76	Gangrenous appendix, with general peritonitis.	96 hours.	Toxæmia.	6th	14.1.31

TABLE V.

Bacteriological Investigations of Exudate.

Case Number.	Initials.	Age.	Condition at Operation.	Duration.	Smear.	Culture.
220	H.Q.	60	Gangrenous appendicitis, general peritonitis, thick pus.	36 hours.	No organisms.	Few colonies of *Bacillus coli communis*.
223	G.	30	Gangrenous appendicitis, general peritonitis, pus.	48 hours.	Short chain streptococci.	Many colonies of *Bacillus coli communis*. No growth streptococci on blood agar.
235	P.R.	7	Gangrenous appendicitis, general peritonitis, sero-pus.	36 hours.	No organisms.	Sterile.
236	T.F.	42	Burst appendiceal abscess, general peritonitis, sero-purulent exudate.	19 hours.	No organisms.	Two colonies of *Bacillus coli communis*.
237	N.	17	Gangrenous appendicitis, general peritonitis, sero-pus.	32 hours.	No organisms.	Sterile.
238	N.V.	24	Burst appendiceal abscess, general peritonitis, sero-purulent exudate.	10 hours.	No organisms.	One colony of *Bacillus coli communis*.

The investigations shown in Table V were made in six of the last cases of general peritonitis patients operated upon, and, as can be seen by the variation in age of patients and duration of the peritonitis, they are fairly representative of the whole series.

The smears showed that the leucocytes in all cases were morphologically intact and stained well.

One cubic centimetre of exudate was cultured in each case and the sample was taken from the general exudate as it appeared when the peritoneum was first opened. It was not taken from the highly contaminated pus or *débris* in immediate contact with the gangrenous appendix. The smears and cultures were made before a finger had been placed in the abdomen.

The object of the investigation was to show the comparative innocuity of the general exudate, not to determine the infectivity of the primary focus.

Conclusion.

Let me conclude with a final exhortation concerning the greatest cause of peritonitis: Abolish the drainage tube wherever possible in the operative treatment of peritonitis, eliminate purgatives and involuntary muscle stimulants and other forms of embarrassment of an inflamed part in the after-treatment, and the mortality from appendicitis, which at present is a disgrace to the medical profession, will fall to a reasonable figure.

Summary.

1. Owing to the nature and distribution of the contents of the peritoneal cavity, it is physically impossible to effect adequate drainage in cases of diffuse peritonitis.

2. Attempted drainage is not necessary when the primary focus has been adequately dealt with.

3. The harmful effects which may follow the use of the drainage tube are described.

4. In the majority of cases it is unnecessary to remove the peritoneal exudate in the operative treatment of peritonitis.

5. The series of cases analysed would indicate that peritonitis in the early stages is not a serious condition if treated on rational lines.

Acknowledgements.

I should like to acknowledge my indebtedness to Dr. Orton, Pathological Registrar of the Alfred Hospital, for his care and trouble in collecting the records of the series of cases tabulated, and to thank the members of the resident staff for their help and cooperation in the after-care of my patients.

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EARLY DIAGNOSIS AND TREATMENT OF UTERINE CANCER.[1]

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The statistics of the Royal Prince Alfred Hospital agree with those of the outside world in placing cancer of the cervix as the more frequent and more lethal type of uterine cancer. It accounted for 873 out of 970 cases of uterine cancer among patients admitted to the hospital from 1883 to the end of 1932. Our remarks will therefore chiefly concern cancer confined to the neck of the womb.

It is now well recognized that the one pathognomonic sign of early cancer of the cervix is abnormal hæmorrhage, but unfortunately it is not so generally recognized that by the time these bleedings attract the attention of the sufferer or her medical adviser, the cancer is already well established and at times deep-seated. To remedy this state of affairs the patient must be encouraged to present herself earlier, and we on our part should make ourselves experienced in the appearance of growth in its earliest stages and further forestall its ravages by making ourselves familiar with those conditions that predispose to the development of it. To acquire this later knowledge we should in the future pay more attention to the minute naked eye appearance of all chronic cervical lesions and should investigate carefully every case of leucorrhœa, as there is always a short initial period of pre-hæmorrhagic inflammatory leucorrhœa in the development of cervical cancer, altogether distinct from the foul and copious discharge which appears once necrotic ulceration has taken place.

In point of fact we must come to realize that the pathology of the chronic infected lesions of the torn cervix are most important in the prophylaxis of cancer and that it is only by a more careful study of these lesions and their treatment that any improvement is likely to take place in the prevention of this deadly malady.

The present position of the cancer problem in this region is that the techniques of the surgical and radiological methods of treatment are now almost perfected and it is useless to expect any further improvement by these means so long as patients continue to present themselves in the late stages of the disease. A study of the world's literature shows that the five-year absolute cures obtained by the present forms of treatment vary from 15·4% to 36·4% in the different clinics, all of which, unfortunately, report a great preponderance of advanced cases (see Table I). However, this is the crux of the

[1] Read at the Fourth Australian Cancer Conference, Canberra, March, 1933.

ILLUSTRATIONS TO THE ARTICLE BY DR. H. H. SCHLINK AND DR. CLEMENT L. CHAPMAN.

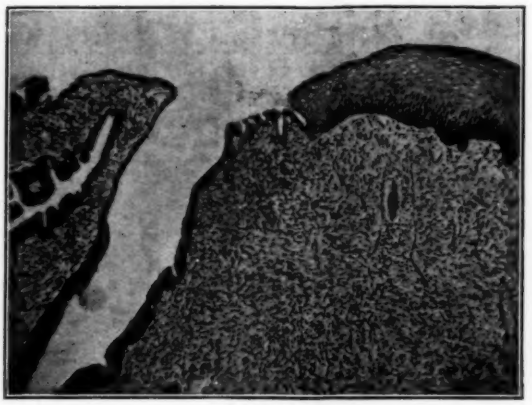

FIGURE I.

Shows the normal, very well defined transition from the squamous epithelium of *portio vaginalis cervicis* to the columnar epithelium of the cervical canal. The so-called transitional zone.

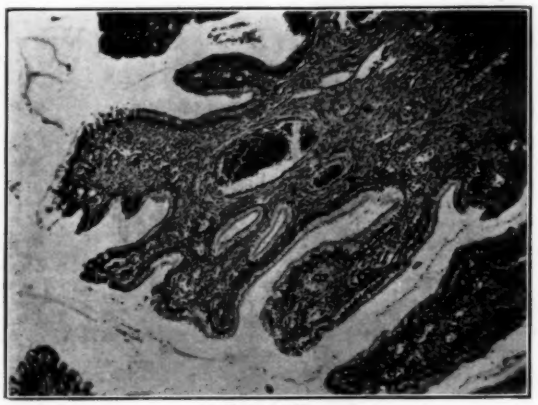

FIGURE II.

Papillary erosion. Columnar-celled epithelium has grown down in papillæ and "eroded" the squamous-celled covering.

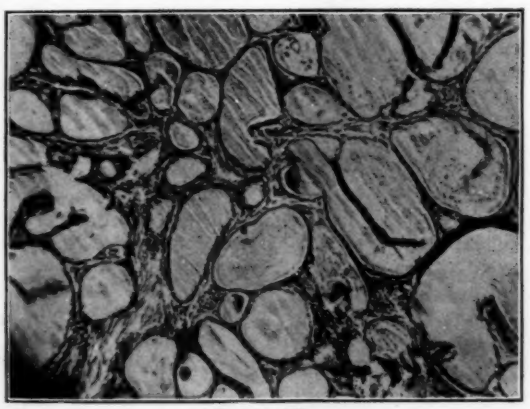

FIGURE III.

Follicular erosion. Cervical glands have become cystic (ovule of Naboth); ducts have become scarred over and occluded.

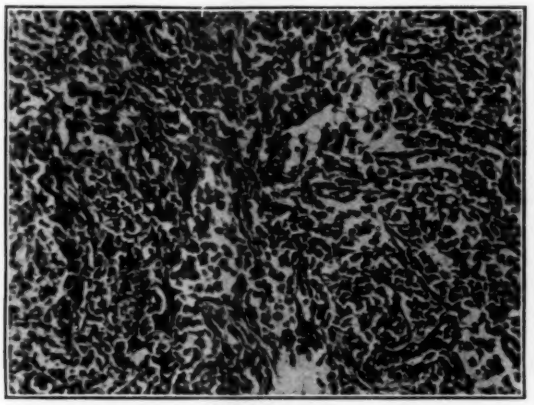

FIGURE IV.

Anaplastic carcinoma of cervix, very cellular, no cell grouping, little fibrous stroma. The so-called unripe type of squamous carcinoma.

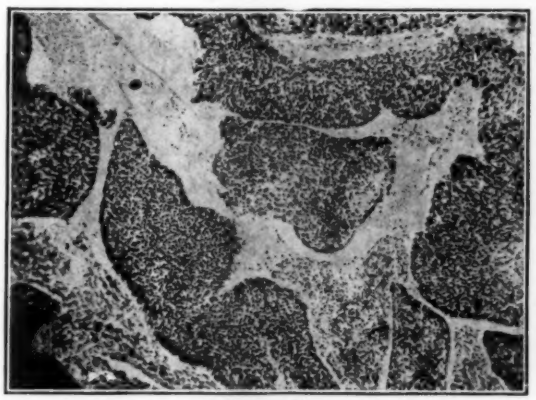

FIGURE V.

Transitional-celled carcinoma of cervix, cells grouped into definite masses, well marked stroma, no keratin formation. The so-called mid-ripe type of squamous carcinoma.

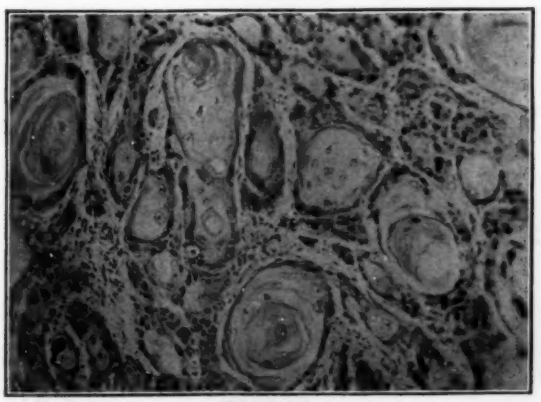

FIGURE VI.

Squamous-celled carcinoma of cervix, well defined cell masses with keratin in centre, stroma well marked. The so-called ripe type of squamous carcinoma.

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FIGURE VII.

Basal-celled type of carcinoma of cervix, masses of small, deeply staining cells, stroma well marked. On left of section is shown hyperplastic proliferation of cells lining an actual cystic follicle.

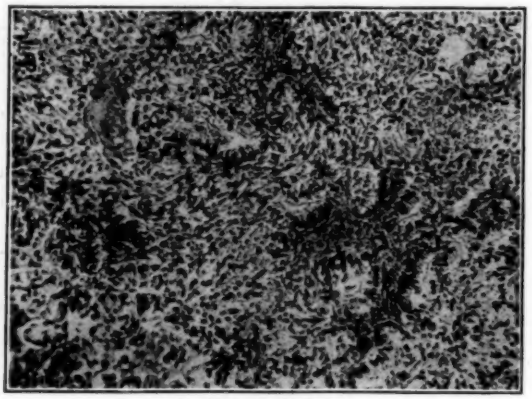

FIGURE VIII.

Adeno-carcinoma of cervix. Columnar cells arranged in alveoli.

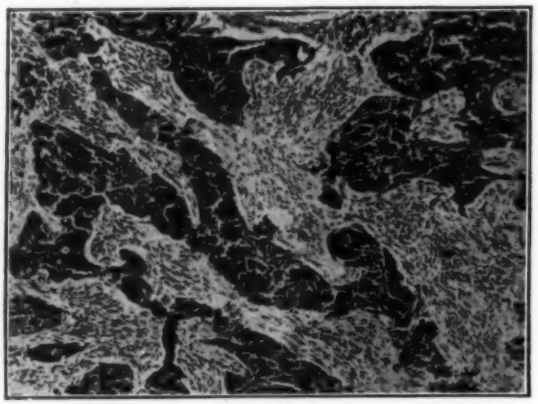

FIGURE IX.

Biopsy of transitional-celled squamous carcinoma of cervix before application of radium.

FIGURE X.

The same growth as Figure IX one week after radium application. Dose, 4,000 milligramme-hours; filtration, one millimetre of platinum. Carcinoma cells show cytoplasm clear, nuclei vacuolated, radium connective tissue reaction, small round-celled infiltration.

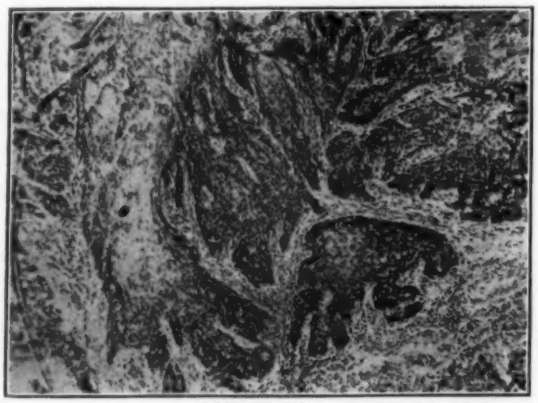

FIGURE XI.

Biopsy of transitional-celled squamous carcinoma of cervix before application of radium.

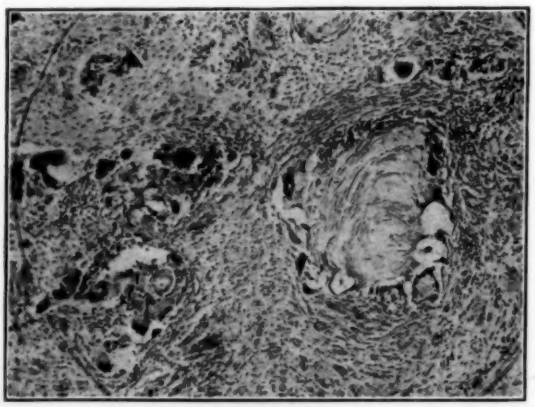

FIGURE XII.

The same growth as Figure XI, showing advanced changes due to radium application four to five weeks afterwards. Carcinoma cell masses replaced by material resembling keratin surrounded by foreign body giant cells, connective tissue reaction marked. Dose, 3,556 milligramme-hours; filtration, one millimetre of platinum.

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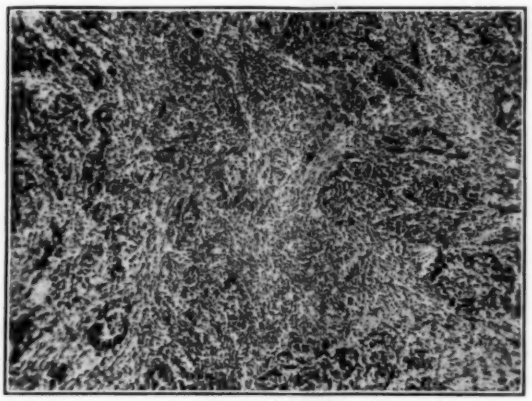

FIGURE XIII.
Biopsy of more diffuse type of transitional-celled carcinoma of cervix before radium application.

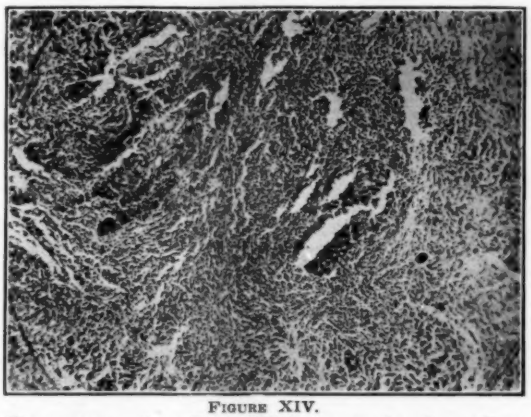

FIGURE XIV.
The same growth as Figure XIII seven weeks after radium application. Carcinoma cells almost entirely replaced by connective tissue, some necrosis in places. In upper left hand corner is a group of carcinoma cells showing advanced destructive changes ("embalmed area"). Dose, 4,992 milligramme-hours; filtration, one millimetre of platinum.

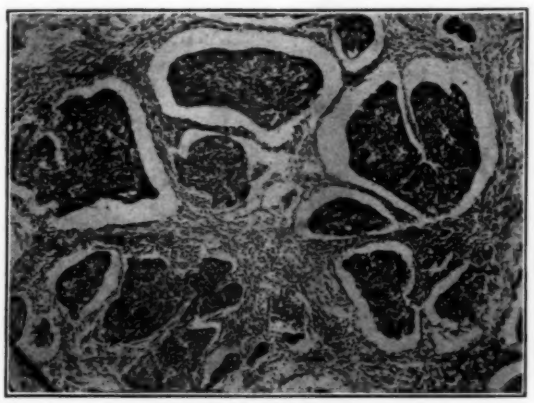

FIGURE XV.
Biopsy of transitional-celled carcinoma of cervix before radium application.

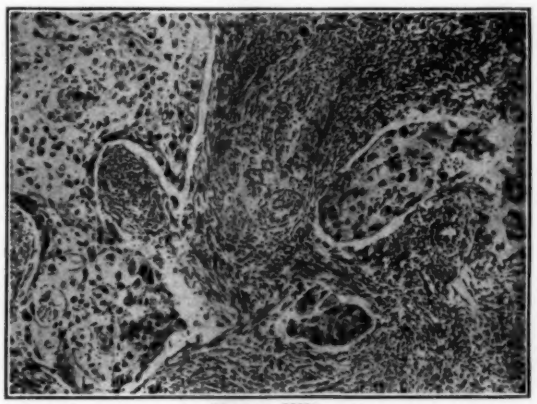

FIGURE XVI.
The same growth as Figure XV four weeks after radium application, 2,500 milligramme-hours, filtration one millimetre of platinum, showing mild changes, cell outlines indistinct, nuclear vacuolation, some connective tissue change. Dosage insufficient.

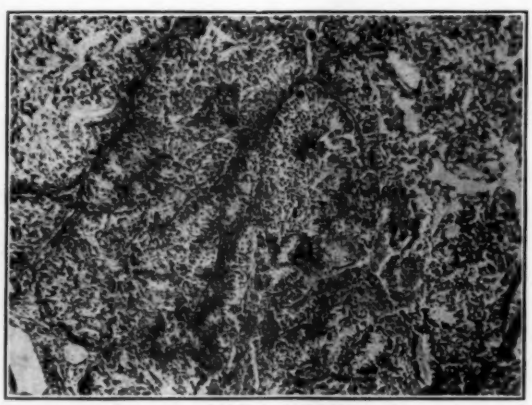

FIGURE XVII.
Biopsy of columnar-celled adeno-carcinoma of cervix. Considerable mucus in cells.

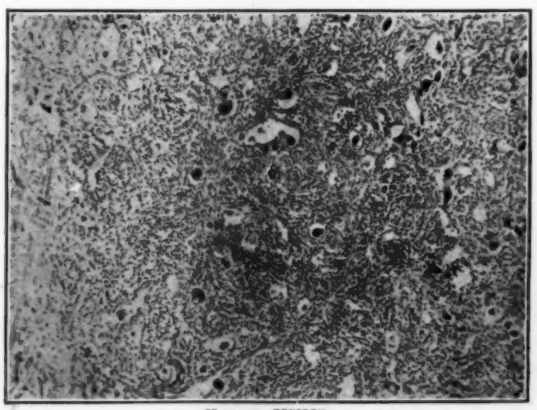

FIGURE XVIII.
Same growth as Figure XVII, showing very complete destruction of growth, a few giant cells alone remaining in a mass of connective tissue, radium reaction. Dose, 6,500 milligramme-hours; filtration, one millimetre of platinum.

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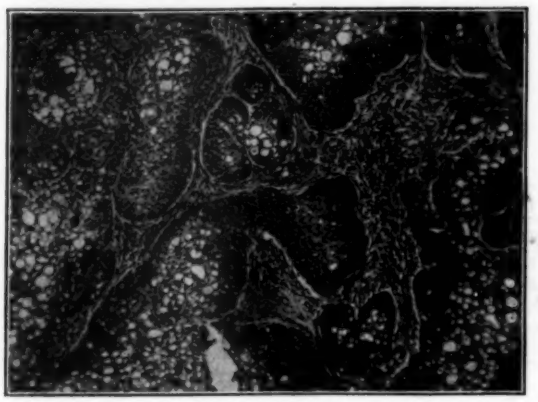

FIGURE XIX.

Biopsy of transitional-celled carcinoma of cervix with some cellular histolysis.

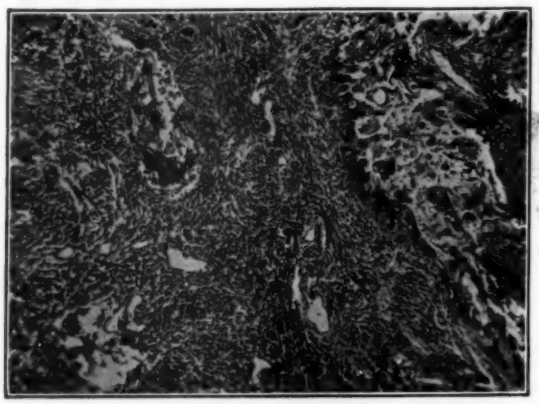

FIGURE XX.

Same growth as Figure XIX after radium, advanced destruction with one or two embalmed areas. Dose, 4,200 milligramme-hours; filtration, one millimetre of platinum.

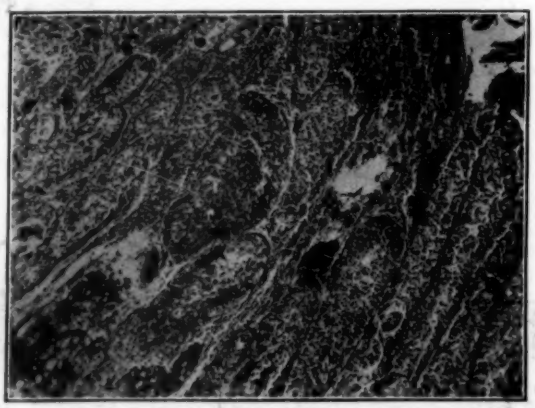

FIGURE XXI.

Lymph gland from broad ligament of same case as Figures XIX and XX, showing marked invasion with cancer cells. In lower left hand corner is original tissue of lymph gland. There is no visible change in the cancer cells of this gland, despite the destruction in the primary growth, shown in Figure XX.

TABLE I.

Five Year Absolute Cure Results (Cancer of Cervix).

Radiation.

Authority.	Number of Cases.	Absolute Cure.
World Literature Franque	6,827	17·45%
Doderlein	1,723	15·4% to 16·5%
(Voltz)	(404)	(24·0%)
Wintz	740	18·9% to 20·9%
Frosell Heyman	502	23·3%
Régaud	678	16·6% to 26·0%
(Lacassagne)	(317)	(33·0%)
Bowing-Fricke	1,094	21·8% to 23·0%
Gray Ward	259	24·7%
Schmitz	486	19·14%
	(156)	(22·43%)

Operation.

World Literature Heyman	5,816	18·56%
Wertheim (abdominal) ..	1,000	18·56%
	(500)	(20·0%)
Bonney (abdominal) ..	538	24·5%
	(133)	(33·8%)
Schauta (vaginal)	696	19·8%
	(253)	(22·5%)

Operation and Radiation.

Franque (abdominal) ..	—	28·1%
Peham (vaginal)	—	28·0%
Adler (vaginal)	—	31·8%

All Methods.

Wilhelminenspital	—	36·4%
Adler	—	36·0%

Absolute cure rate varies from 15·4% to 36·4%.

Relative Cure Rate.

Group 1. Operable.	Group 2. Borderline.	Group 3. Inoperable.	Group 4. Incurable.
75% to 90%	35% to 50%	10% to 20%	Zero

situation, as both surgeons and radiologists claim very similar relative cure rates:

Group 1, or early localized cancer, about 75% to 90%.
Group 2, or the doubtfully localized, 35% to 50%.
Group 3, or the cases with involvement of the parametria and glands, 10% to 20%.
Group 4, or those with fixed or frozen pelves accompanied by metastases, local or distant, zero.

It is obvious that the poor absolute end results at present obtained are due to delay in treatment and the difficulties of early diagnosis. We must therefore recognize that any improvement in the control of cancer depends solely on prophylaxis, earlier diagnosis and prompter treatment. Thus we should first bend our energies more and more on the study and care of all those cervical lesions which might be the forerunners of malignant growths, perfect our art of diagnosis when the growth first appears, and propagandize the women of this country to present themselves for examination at the menopause and especially on the first sign of any reappearance of bleeding after that age has been passed. Out of 970 cases among patients admitted to the Royal Prince Alfred Hospital since 1883, 706 occurred after the menopause.

TABLE II.

Predisposing Lesions and Prophylactic Data.

Predisposing Diseases.

1. Papillary erosion.
2. Follicular erosion.
3. Leukoplakia.
4. Desquamated areas.
5. Precancerous coating.
6. Infected lacerations.
7. Polyps and papules.
8. Endocervicitis and cervicitis.
9. Residual stumps.
10. Myopathic uteri.
11. Fibromyomata *et cetera*.

Colposcopic Examinations.

1. Normal transition zone	20%
2. Normal but misshapen transition zone—ectropion	10%
3. Abnormal transition zone leukoplakia nodules and ulcers	70%

2% of these are precancerous.

Cervical Operations and Residual Cervices.

Authority.	Number of Cases.	Treatment.	Follow Up.
Bossi	7,000	Low amputation.	Nil
Hunner	2,895	Amputation or electrocautery.	Nil.
Pemberton and Smith	3,814	Trachelorrhaphy.	5 cancer.
	740	Amputation.	Nil.
	1,406	Electrocautery.	Nil.

Per contra, 669 patients with proven cancer had had only 12 plastic operations.

Residual Stumps that Developed Cancer.

Authority.	Number of Cases.
Herbert Spencer (1906)	28
Polak (1912)	250
Sanders (1924)	102
Fleishmann (1925)	50
Jeanessey and Chavaumez	85
Branscombe (1920)	46
Gray Ward (1932)	20
Mayo Clinic	13
Royal Prince Alfred Hospital	35
Total	629

Prophylaxis.

With regard to prophylaxis, it is reasonable to assume that cancer does not start from healthy tissue. However, it must start in some spot the cells of which were at one time non-cancerous, and as no specific organism has yet been found, we cannot be far from the truth if we blame the chronic irritation of a superimposed cervicitis as the real urge to the malignant metamorphosis. Thus the radical treatment of chronic cervical lesions is the real prophylaxis of cancer in this part of the genital tract. This is supported by the work of Bossi (1913), who did a low amputation for diseased portions of the cervix in 7,000 cases and did not find a single case in which cancer developed after many years of follow-up (see Table II). The statistics of Pemberton and Smith (Graves's clinic, Boston) also show the prophylactic value of cervical surgery. During a period of fifty-two years, 5,962 patients with chronic cervicitis were treated, 3,814 by trachelorrhaphy, 740 by amputation, and 1,406 by electro-cautery. In many of the cases (2·39%) unsuspected cancer was discovered in the removed tissue. Only five women out of the 5,962 operated

upon developed cancer subsequently, and they all had had trachelorrhaphy in which probably the whole of the eroded tissue was not removed. This goes to prove that amputation and electro-cautery are superior forms of treatment, and of the two amputation is the better.

Per contra, they found that in 669 cases of proven cancer of the cervix only twelve patients had had a cervical plastic operation. Hunner's statistics support the preceding figures; out of 2,895 patients with chronic cervicitis subjected to either amputation or cauterization, not a single patient had developed cancer ten years later.

All will agree that these experiences strongly suggest not only the causative factor of cancer of the cervix, but also the prophylactic value of attending to all chronic lesions of the cervix, among the commonest of which are the so-called erosions or cervical ulcerations of the older writers. Most of these occur in parous women who have suffered a cervical laceration which has become infected by specific or non-specific organisms. Pathology divides them into two types: (i) The papillary type (Figure II), which results from an inflammatory subepithelial exudate; the exudate causes the pavement epithelium to be desquamated and the papillary processes to be exposed. (ii) The cystic nodular type (Figure III), in which the exposed ducts of the underlying racemose glands are healed over or blocked, the typical Nabothian follicles being caused. This second type of erosion might in certain instances be regarded as the late stage of the papillary variety of erosion in its attempt to heal. The treatment of these two types of erosion is entirely different. The first, if mild, can be healed by escharotics or, more efficiently, by ironing over with diathermy. The second can be cured only by amputation.

As out of 970 cases of uterine cancer 849 occurred in parous women, it is good prophylactic treatment that all cervical lesions which have not been attended to at confinement should be rectified as soon as possible. Inflammations of the endocervix should be efficiently treated, and, if they become intractable, enucleated. When the inflammation extends to the stroma, causing diffuse cervicitis with myometrial extension resulting in fibrosis, it might call for hysterectomy with endocervical enucleation.

We would here make a strong plea for the abandonment of the classical subtotal hysterectomy and the substitution of the total operation, or preferably the safer and less difficult subtotal operation with enucleation of the endocervix, as described in *The Journal of the College of Surgeons of Australasia*, Volume I, July, 1928, page 103. The reason for this plea is the high percentage of residual cervices which subsequently become cancerous when the older classical subtotal hysterectomy is used. We have already had 35 since 1910 in our hospital, and the following are some of the figures from other parts of the world:

1. Polak (1921), 250 cases of cancer of the stump, all of which recurred after one year.
2. Sanders (1924), 102 cases.
3. Fleishman (1925), 50 cases.
4. Jeanessey Chavaumez, 85 cases.
5. Branscombe (1930), 46 cases.
6. Grey Ward (1932), 20 cases.
7. Mayo Clinic, 13 cases.

As long ago as 1906 Dr. Herbert Spencer reported 28 cases.

We should like to record that we have performed close on one thousand subtotal hysterectomies with enucleation of the endocervix for various diseases of the uterus and have met with only one case in which the residual cervical shell was the site of subsequent cancer. This was one of our early cases, in which we probably had not removed the entire transitional zone. We should be pleased if anyone meeting subsequent cancer in our enucleation cases would kindly inform us of the fact.

Such are some of the measures that will prevent the development of uterine cancer.

Early Diagnosis.

Now, as regards the diagnosis of early cancer of the cervix, the problem is not so easy, as there are in the beginning no symptoms, and ordinary inspection and palpation are not at all conclusive. However, the introduction of the colposcope by Professor H. Hinselmann, of Hamburg, will in the near future throw a new light on the study of erosion, leucoplakia, and the so-called carcinomatous surface coating of the cervix. We are at present making ourselves familiar with the technique of this new eye, which gives a stereoscopic view of the cervix enlarged tenfold, and by a special construction enlargement up to fortyfold. It is enlightening to record Hinselmann's observations in a discussion on the use of this instrument in the diagnosis of cervical lesions.

1. Normally the vaginal mucosa should end sharply and ring-like at the external os. This is seen in about 20% of colposcopic examinations. Cancer never or hardly ever occurs in such cervices.

2. An ectropion of the columnar-celled mucosa of the cervical canal does not predispose to carcinoma if the normal relations between vaginal and canal mucosa are maintained, even if it is misshapen. Ectropion is usually due to a laceration without subsequent infection. They are observed in about 10% of such examinations.

3. If a cervical erosion becomes chronic or heals, then a transition zone between the vaginal and canal mucous membrane forms, in which nodules or desquamations may be seen. Such changes may become or are atypical and hence precursors of carcinoma. Such transition zones are present in about 70% of such examinations.

The atypical changes in the transition zones are either composed of keratinized surface epithelial cells termed leucoplakia, or are desquamated areas devoid of pavement epithelium and may be recognized by Schiller's iodine test. Of the leucoplakias or ulcerations discovered in routine examination Hinselmann considered 2% precancerous.

Whenever there is any doubt as to the atypical character of the transition zone, there should be no delay or time wasted in keeping the patient under observation, as is so often done, but a biopsy should be taken wide of the eroded or leucoplakic patch and submitted to microscopic examination. In very suspicious cases the cervix should be amputated forthwith.

When cancer commences inside the cervical canal or in the uterine cavity, the diagnosis can be made only by contact bleeding or a thorough dilatation and curettage with microscopic examination of the scrapings.

Histological Classification.

Before proceeding to the discussion of treatment it might be as well to demonstrate the histological structure of the various types of uterine cancer as found in the biopsies of our cases (see Table III).

Table III.

Histological Classification.

(a) Precancerous (squamous or columnar)
(b) Squamous epithelioma—619

	Healy
(1) Keratinized or ripe	17%
(2) Transitional or midripe	62%
(3) Anaplastic or unripe	21%

There is a fourth variety resembling the basal cell carcinomata of skin.
(c) Adeno-carcinoma—

(1) Adeno-carcinoma of cervix	32
(2) Adeno-carcinoma of body	71

There is a rare type of uterine cancer that shows columnar and squamous mixed.
(d) Chorio-epithelioma, 1; Fibro-sarcoma, 5; Endometriomata *et cetera*.

Precancerous Tissue.

We are on the look-out for slides that will show tissues which are half way between the orderly cell arrangement of normal tissue and the disorderly distribution and invasion of surrounding tissues by frankly malignant cells. The precancerous change should be indicated by a variation in size of the cells and hyperchromatic changes in the nuclei. The cells should be, as it were, getting ready to take on their task of invasion. We have found one or two cases which need further study before they are presented.

Squamous Epitheliomata (Cervix).

It is usual to divide squamous epitheliomata of the cervix histologically into three classes:

	Healy.
(a) Anaplastic or unripe	21% (Figure IV).
(b) Transitional or mid-ripe	62% (Figure V).
(c) Keratinized or ripe	17% (Figure VI).

The anaplastic type show diffuse invasion of tissues by large epithelial cells devoid of any orderly arrangement. The transitional variety show irregular clumps and columns of cells with hyperchromatic bizarre nuclei, a few prickle cells, but no keratinization. The masses are well defined as compared with the anaplastic variety. The keratinized cell variety shows a more regularly arranged mature cell with well marked prickles and epithelial pearls. There is a fourth variety (Figure VII) occasionally met with which resembles the basal-celled epithelioma of the skin; the cells are small and compact and the nuclei stain very deeply.

Although most authorities regard these varieties as indicating the age or ripeness of the whole growth, we have found all varieties in one and the same growth, and it is therefore reasonable to suppose that the classification may not be justified and that the differences found may be due to the particular part of the growth from which the biopsy has been taken. It is reasonable to suppose that the centre of the growth would be older (and therefore show keratin) than the invading periphery. This is borne out in cases of irradiated uteri which have been removed by operation. The biopsy often shows the anaplastic or mixed variety, and the section from the neoplasm well marked keratinization, because the pathologist has been able to cut his sections from the very centre of the growth.

As regards adeno-carcinomata (Figure VIII), this type of growth, whether originating in the cervix or the body of the uterus, displays the same histological characteristics. The number of the glands increases, encroaching upon the interstitial tissue, the cells multiply and heap up, forming papillomata, as it were, in the duct of the gland, and eventually they break through the basement membrane and invade the surrounding tissue. Occasionally in adeno-carcinoma of the body foci of squamous epithelioid cells are found. Their origin is doubtful. Some authorities hold that they migrate from the cervix, but Aschoff and others maintain that the cancer cell is capable of developing in different directions, producing either a columnar or squamous variety of cells. As we are limited to time, we shall not attempt to discuss chorio-epithelioma, fibro-sarcoma, malignant polypi or endometriomata.

Clinical Classification.

In nearly all clinics Döderlein's four groups for classifying the stage of malignant growth clinically are now used. They have been recognized and adopted by the Radiological Commission of the Hygiene Section of the League of Nations as a basis for statistical investigations in this field of cancer work.

Cases from the year 1910 to 1932 inclusive:

Group 1. Operable cases	120
Group 2. Border-line cases	164
Group 3. Inoperable cases	391
Group 4. Incurable or very advanced cases	53
Total	728

Treatment.

In 1930 Dr. Sandes, the Director of Cancer Treatment, requested the gynæcologists of the metropolitan hospitals of Sydney to meet and to decide on some plan of attack on uterine cancer. As a result of that conference it was decided that for the next five years the Royal Prince Alfred Hospital should adopt, as far as possible, the combined treatment of radium and operation; the Sydney Hospital, radium alone; and Saint Vincent's Hospital, combined radium and X rays. Uniform records were to be kept in specially designed books supplied by the Cancer Research Committee, and the conference

Table IV.

Correlation of the Histological and Clinical Signs and Treatment (after Schmitz).

Group.	Macroscopic Appearance.	Histological Development.	The Grouping of the Extent of the Growth.	The Symptoms.	Treatment.	Average Five Year Cure Rate.
1	Nodule.	Cellular atypia.	Clearly localised growth. Normal mobility of uterus.	None.	Radium + operation.	75% to 90%
2	Ulcer or papilloma.	Invasion of basement membrane.	Doubtfully localised. Impeded mobility of uterus.	Contact bleeding.	Radium + operation.	35% to 50%
3	Large ulcer or cauliflower growth with necrosis.	Destructive tendencies.	Invasion of parametria or regional lymph glands. The entire tumour mass is mobile.	Haemorrhage, discharge.	Radium + operation or radium only.	15% to 20%
4	Crater.	Metastasis, local or distant.	1. Frozen pelvis with absolute fixation of tumour. 2. Local dissemination to vagina, bladder or rectum. 3. Distant metastases.	Haemorrhage, discharge, pain.	Local spooning and zinc chloride.	Zero.

was to meet from time to time to report progress. The present paper is based on this short experience of the combined method as practised by us at the Royal Prince Alfred Hospital, and although seven more years will have to elapse before we can give our absolute and relative five-year cures for the whole of this present series, we have already been able to provide interesting data concerning the irradiated tissues we have removed, as well as data for determining by result the dosage needed for the various types of growth.

The following is a summary of the routine treatment of a case of uterine cancer adopted by us:

1. History with duration of signs and symptoms.
2. Physical examination with detailed descriptions of growth.
3. Wassermann test and blood count.
4. Biopsy and diagnostic curettage.
5. Insertion of radium.
6. Description of the local result of radium on the growth.
7. Operation.
8. Detailed description of removed organs.
9. Histological examination of the removed irradiated tissues which are contrasted with the histology of the original biopsy.
10. Follow-up.

Pre-Operative Use of Radium.

We have found that the use of radium usually reduces the size of the growth and helps any adventitious inflammatory induration to disappear, thereby making movable a growth that was originally fixed. Thus the use of radium rendered many border-line and some apparently inoperable growths operable. From examination of the removed uteri we discovered that all growths that have not broken through their uterine shell are easily extirpated. But, above all, we learnt that radium eradicated sepsis and is, from the operative point of view, the best surgical antiseptic yet met with. We believe that it is this characteristic of radium which has allowed us to do the last fifty radical Wertheim operations with no mortality and little or no morbidity.

From a study of the records of uterine cancer since the inception of the hospital (970 cases) we discovered that most of the post-operative deaths not due to injury to the ureters or bladder were caused by septic peritonitis, probably due in most cases to infection from the necrotic growth. Radium *plus* Bonney's blue has almost eliminated this risk.

Application and Dosage: In most cases we have found it advisable to curette away all the necrosing *débris* before inserting the radium. We feel certain that such treatment, if done gently and intelligently, has no influence on the spread of the disease, as in these advanced cases there is always a strong adventitious protecting zone, the lymphatics and veins of which are blocked either by growth or thrombosis. For the same reasons we do not think the careful excision of the biopsy specimen with the cold knife tends to increase the risk of extension, if the cut is made either wide through the healthy tissue or well within the margin of the growth. We irradiate the full length of the uterus by inserting into the uterus 25 to 50 milligrammes of radium element (one millimetre of platinum) in a rubber-covered tube and a plaque (lead box), or corks of 20 to 30 milligrammes (one millimetre of platinum). If more suitable, we insert needles amounting to 20 milligrammes (one millimetre of platinum) instead of plaque (lead box). Lately we have abandoned the plaque (lead box), corks or other containers for Donaldson's method of needles, as in many cases we found it difficult to retain the former in constant accurate position. We remove the plaque (lead box) or corks, when used, after fifty hours for fear of burning the bladder or rectal mucosa, but leave the tube in the uterine canal up to one hundred hours, provided there is a good shell of uterine tissue present. The combined dosage usually amounts to between 4,000 and 5,000 milligramme-hours with a screenage of one millimetre of platinum.

Operation.

Although we have operated on patients from one week to seven weeks after the application of radium in order to study its effect at varying intervals of time, we would advise that the operation be undertaken not later than three or four weeks after the application. Before this time the tissues are swollen and œdematous and much bleeding is met with, while, if a longer time is allowed to elapse, the ureters are very hard to isolate, as the tissues become dry, cicatrized and firmly bound down, or in other cases chronically swollen and indurated. Our experience has been that, if done between the third and fourth week after the application, the operation is comparatively easy and the bleeding much less than in a radical operation that has not been preceded by radium treatment. The radium seems to exert an effect on the pelvic vessels, especially the veins, which appear smaller and do not bleed so much when cut across or broken.

As regards operability after radium, whenever the uterus can be freely moved with parametria pliable and no obvious fixation to the bladder or rectum, the case is further investigated by laparotomy to decide whether removal of the growth is possible. Extension on to the vaginal walls is no contra-indication to operation, provided the extension is superficial. It is unnecessary to add that patients about to be irradiated and operated upon should be rendered as fit as possible beforehand. Thus, if they are very anæmic a transfusion should be given, and if they are syphilitic a short, intensive course of "Salvarsan" and mercury should be undertaken.

Without a thorough understanding of the action of radium on uterine cancer, which can be gained only by a histological study of the irradiated tissues, radium treatment can be empirical only. Our combined method has the virtue of supplying this necessary histological material, and as a scientific check will, we are sure, eventually lead to regulation of dosage and appropriate methods of application.

In order that you might more clearly appreciate the sections about to be shown you, we will briefly enumerate the changes that were observed in irradiated cervical cancer by Canti and Donaldson (see Table V).

Three months after exposure embalmed areas were still present. The unanswered question is: Are these embalmed areas capable of initiating a new spread of the disease?

We adopt the following routine of work in our department at the Royal Prince Alfred Hospital. The removed uterus, as well as the parametrial tissue and any enlarged glands, are subjected to thorough histological examination in our presence. The original biopsy is first shown, next the irradiated tissues, and thirdly the specimen is gone over with the pathologist to ascertain where the sections have been taken from (Figures IX to XXI).

We publish herewith typical sections indicating the effect of minimum and maximum doses on different types of growth at varying intervals of time; they will convey the conclusions we have arrived at up to the present.

Conclusions.

1. Radium as applied by us had no effect on cancer deposited in the lymph glands, which were secondarily affected in 10% of our operative cases.
2. Our best results were with dosage of 4,000 to 5,000 milligramme-hours (one millimetre of platinum).
3. Dosage of 2,000 milligramme-hours is not enough to eradicate even a localized growth.
4. Vaginal dosage should never exceed 2,000 milligramme-hours for fear of late injury to the bladder or rectum.
5. The best results were obtained when the whole of the uterine cavity was irradiated.
6. In some cases we prefer needles to vaginal corks or plaques (lead boxes), because the latter are too easily displaced.
7. We prefer to operate not before three weeks following exposure nor after four weeks.
8. We cannot yet make up our minds whether the embalmed cells and other disintegrating cancer cells are going or returning.
9. We have abandoned diathermy on account of the frequency of subsequent rectal, vesical and peri-

TABLE V.

Result of Radium on Cervical Canal.

Observation by Canti and Donaldson.	Our Own Irradiated Tissues.
1. *For two days after exposure:* Cessation of mitosis.	1. Original biopsy and irradiated tissue one week after exposure (4,000 milligramme-hours) (Burnett). (Figures IX and X.)
2. *Third and fourth day after exposure:* (a) *Tissue nearest to radium:* Necrosis (no nuclear staining). (b) *Middle zone:* Return of mitosis (increased and abnormal) with droplets in cytoplasm. (c) *Distant zone:* Cells and nuclei increased in size with abnormal mitosis.	2. Original biopsy and irradiated tissue four to five weeks after exposure (3,550 milligramme-hours) (Reader). (Figures XI and XII.)
3. *Seven or eight days after exposure:* All mitosis has ceased. Nuclei irregular in shape and staining. Cytoplasm vacuolated. Leucocytic invasion started.	3. Original biopsy and irradiated tissue seven weeks after exposure (4,992 milligramme-hours) (Taylor). (Figures XIII and XIV.)
4. *Six or eight weeks after exposure:* Only trace of growth are embalmed areas. Concentrically laminated bodies take place of former strands of growth. Progressive fibrosis (secondary) has set in.	4. Original biopsy and underdose of radium four weeks after exposure (2,500 milligramme-hours) (Ettrich). (Figures XV and XVI.)
5. *Three months after exposure:* Embalmed areas still present.	5. Original biopsy and large dose of radium three weeks after exposure (6,500 milligramme-hours) (Jones). (Figures XVII and XVIII.)
	6. Original biopsy and disappearance of all signs of growth in cervix, but persistence in lymph glands three weeks after exposure (4,200 milligramme-hours) (Johnston). (Figures XIX, XX and XXI.)

toneal fistulæ, ten of which are recorded in the Royal Prince Alfred Hospital records.

10. Most of our biopsies are of the transitional or mid-ripe variety. Many of these were changed after the radium to the ripe or keratinized variety. This may be due to the section being taken from the centre of the growth, as the pathologist has the whole uterus.

11. We have not yet made up our minds which, the anaplastic (unripe) or the keratinized (ripe), is the more radio-sensitive or radio-resistant.

12. We have found that many growths display unripe, mid-ripe and ripe characteristics, and that even an adeno-carcinoma of the body will show foci of squamous cells. Whether these originated in the cervix or arose from the fact that the epithelium that initiates the growth can differentiate into columnar or squamous cells at will must be left for the future to say.

Inoperable and Very Advanced Cases.

Any cases that were unsuitable for the combined method were treated with radium alone, the dosage being determined by the experience we had gained from the irradiated tissues of the operated cases. As Sydney and Saint Vincent's Hospitals will report fully on this aspect of the question, we shall confine ourselves to a study of the combined method in this paper. We shall record all our personal results in a subsequent paper. However, as regards the very advanced cases, we are coming to the conclusion that radium only makes them worse and causes the patients to die with a terrific amount of pain and agony. Among the 210 patients treated by radium alone at the Royal Prince Alfred Hospital there were nine immediate deaths (4·28%). Most of these were in the so-called frozen pelves cases (Group 4). Two patients died from hydronephrosis due to pressure of the resulting radium induration on the ureters, and they indicate the danger of using radium in these advanced cases. In dealing with these cases we have come to the conclusion that it is better to curette away the necrosing tissue and to apply zinc chloride or acetone, as the older gynæcologists did. This relieves the patient of discharge and hæmorrhage. Once a growth is firmly fixed to the pelvic walls, rectum and bladder, the frozen pelvis, as it is called, radium and X rays should not, in our opinion, be used.

Follow-Up.

We have started a follow-up system in which the patient is seen each month for the first six months. After that she is seen each three months for the next two years, and then each six months until the seventh year is passed. We have seen recurrences in this hospital at the seventh year and feel that a patient is not absolutely safe from such a happening until the tenth year is passed.

The Combined Method of Treatment.

As far as we can discover from the world's literature, the best absolute five-year cure rate is obtained by those workers who have recourse to all methods of attack: irradiation *plus* operation, operation *plus* irradiation, or radiation alone. For example, Wilhelminenspital reports 36·4% and Adler 36% of cures. We personally are of opinion that radium should occupy the same place in the operative treatment of cancer as "Salvarsan" does to mercury and iodides in the treatment of syphilis. Radium acts quickly, kills the local lesion or renders it inert, and thereby gives time for carrying out operative measures in a clean field. This antiseptic property cannot be over-estimated by the surgeon. We further believe that the uterus should be removed after the use of radium, whenever possible, as it is impossible to rehabilitate a fifty-year-old uterus, which must always remain an organ subject to age period degenerations and in many instances becomes the seat of post-radial pyometra. Lastly, we have observed that the average cervical cancer takes four years to kill the patient, even if untreated, and we therefore believe that the ten-year absolute cure statistics of radium alone will be far below those of radium *plus* operation.

Acknowledgements.

Before finishing we should like to thank Dr. Sandes, the Director of Cancer Treatment of the University of Sydney, for his encouragement and helpful suggestions, and Dr. A. H. Tebbutt and Dr. Geoffery Davies for their interest and help in the histological work connected with this investigation.

DENTAL HEALTH EDUCATION IN NEW SOUTH WALES.

By E. Brangwin Green,
Chairman of the Dental Health Education Department of the Australian Dental Association.

Of late years the medical and dental professions have been more closely brought together, largely because the relationship which exists between dental affections and general systemic diseases has been more clearly recognized.

It is now realized that the closest cooperation between medical and dental practitioners is essential to the successful treatment of disease.

The Australian Dental Association holds the opinion that preventive dentistry is more important to the community than reparative dentistry, and so strongly does it hold this view that the New South Wales Branch of the Association has, at considerable expense, established a Dental Health Education Department as a definite branch of its activities.

The object of this article is to bring the aims and objects of this department under the notice of the medical profession in an endeavour to arouse their interest and enlist their sympathetic cooperation.

The medical profession takes pride in the fact that year by year its activities are increasingly being directed to the field of preventive medicine,

in spite of the fact that the more successful these activities become, the less work will the general practitioner have to do. Similarly, the dental profession is devoting more attention to preventive dentistry and dental hygiene. Surely the efforts of the two professions should go hand in hand.

The Dental Health Education Department of the Australian Dental Association is endeavouring to carry out its aims by disseminating its propaganda through many and varied channels. It recognizes the great importance of educating the young members of the community, while not forgetting those of more mature years and judgement.

Some of the results already achieved may be briefly mentioned:

1. For fifteen months weekly broadcast talks were given from 2FC and 2BL stations. These have now been temporarily discontinued, but it is hoped to give a new series shortly.

2. A series of articles has been published in the daily Press.

3. An essay competition and examination in the State schools has just been successfully carried out and prizes to the extent of £30 have been given. In this effort the Public Education Department kindly cooperated through the headmasters of the various schools. The Association considers this a most important method of educating the children, and intends to promote a similar competition annually. It is hoped that ultimately it will embrace all schools, State and private.

4. Talks are being given at regular intervals to the mothers at the various free kindergartens.

5. Articles are being published from time to time in various journals and magazines, including those published by large firms and by the Retail Traders' Association, such as the *Hordernian, Dajonian, Retail Traders' Association Journal et cetera.*

6. A series of twelve dental talks has been compiled and distributed through various firms allied to dental practice.

7. Articles for very small children are being published regularly in the *Junior Telegraph.*

In addition to the results already achieved, the department has in view the following activities:

1. Talks to the mothers at pre-natal clinics. These will be carried out with the endorsement and approval of Dr. S. E. Morris, Director of Maternal Welfare. This is considered a very important form of education, especially in regard to the diet of the expectant mother.

2. Distribution of literature, education of officers, and occasional talks to the members of each troop of Boy Scouts and Girl Guides.

3. The publication of articles in the various journals of the two above-mentioned organizations.

4. A second school competition next year, if possible, on a wider basis and embracing all schools in the State. It is hoped to take in the Great Public Schools and the corresponding type of girls' schools.

5. The organization of lectures by local dentists to the members of the parents' and citizens' associations connected with the schools throughout the State.

6. The encouragement of dental attention to children. Special efforts are to be directed towards building up resistance to dental disease and to establishing the proper growth and development of the teeth and jaws. In this connexion representations will be made to the proper authorities, whenever possible, pointing out the urgent need for the establishment of a central dental clinic for children. With the exception of the dental clinic at the Royal Alexandra Hospital for Children, there is no provision in Sydney for free dental treatment for children. The need for such a clinic is recognized by all organizations dealing with the young.

7. Efforts are being made to interest the life insurance companies and to secure their help in distributing leaflets *et cetera.* Already one large company has offered space for articles in its monthly journal.

It will be seen from the foregoing that the programme mapped out by the Dental Health Education Department of the Australian Dental Association is a fairly comprehensive and ambitious one.

It is hoped that they will be assisted in their efforts by the members of the medical profession, because preventive dentistry is, after all, only one branch of preventive medicine.

Assistance of the Medical Profession.

It may be asked in what way can the medical profession help.

Firstly, it can stimulate interest in the importance of dental health as an aid to general bodily fitness. This can be done not only through patients, but through friends and acquaintances, and in the family circle.

Secondly, medical men can help and cooperate with family dentists in educating their patients. This cooperation would also include consultations, if necessary, with dental practitioners in obscure cases of disease.

Thirdly, the latest views on dental health, where applicable, can be embodied in papers read before scientific meetings.

Fourthly, the holding of joint meetings occasionally, of medical men and dentists where problems of hygiene and preventive medicine can be discussed. Now that the Australian Dental Association is firmly established, with branches in all the States and a journal of its own, it is felt that the time has come when cooperation between it and the British Medical Association will be of great assistance in dealing with many of the problems which confront both professions. Of these problems preventive medicine and dental health education are important, and are subjects in which the two professions of medicine and dentistry could well combine.

Reports of Cases.

CLOSURE OF A DEFECT IN THE HARD PALATE BY A PEDICLED TUBE GRAFT.[1]

By G. H. Burnell, M.D., B.S. (Adelaide), F.R.C.S. (Edin.), F.R.A.C.S.,
Honorary Clinical Assistant to the Surgical Section, Adelaide Hospital.

In the case here reported a defect in the hard palate has been closed by means of a pedicled tube graft.

The patient, a boy aged 18 years, had had numerous operations for the repair of a large defect in the hard palate. When first seen, almost the whole of the hard palate was missing and there was no tissue available for its repair. It was then decided to utilize a skin graft; this was after consultation with the staff of the Dental Hospital, who had decided that he was not a suitable patient for an obturator. The tube graft was made from the tissues over the right sterno-mastoid muscle, running from the back of the ear to the right sterno-clavicular joint. At the second stage, the lower end of the tube was divided and implanted into an incision just beneath the chin, where the scar would not be visible later on (Figure I). At the third stage, the upper end of the tube was divided and implanted into the back half of the palatal defect, which had been freshened before-hand (Figure II). A dental block was fitted to keep the patient's mouth partly open, so that he should not bite the graft in his sleep (Figure III). At the fourth stage, the graft

Figure I.

Figure II.

Figure III.

was divided near the anterior end of the palatal defect and sutured into position.

There is firm union around almost the whole of the defect.

Should I have to operate on a similar patient, I would take the graft from the chest below the clavicle, as the scar on the neck is rather unsightly.[1]

[1] One patient herein described was shown at a meeting of the Section of Surgery of the South Australian Branch of the British Medical Association on March 8, 1933.

[1] Since the above was written a second patient has been operated on with complete success from a functional point of view, the graft being taken from just below the clavicle.

Reviews.

MINOR AILMENTS.

Since the first appearance in 1906 "Minor Maladies", by Dr. Leonard Williams, has retained a very wide and deserved popularity. It ought to be read by every junior general practitioner, since it fills many of the unavoidable vacuoles in his training. Subsequent editions have been subjected to revision, omission and substitution to keep in line with the changing thought and trend of modern practice. In the latest edition, the sixth,[2] the chapter on "Dyspepsia" has been largely rewritten, that on "Goutiness" omitted and one on "Salient Symptoms" put in its place, while the last chapter is the work of Dr. I. G. Cobb and deals with "Some Drugs and their Uses". Dr. Williams's literary style is too well known for comment. Never dull, he is often amusing and his dogmatic method of presenting his views will inspire the tiro with a measure of assurance at a period in his career when, possibly, he is all too aware of his own lack of experience and of the quizzical eyes of his patients and their friends. He may occasionally doubt the doctrine declaimed, but seldom the practice prescribed so far as the actual treatment of disease is concerned. On some subjects Dr. Williams holds extreme views and when regarding them his perception becomes monocular. Smoking, he tells us, is a "stupid, and dangerous, and dirty habit" which should cease "on or before the forty-fifth birthday"; those with red hair and those with "facial asymmetry" (are not all faces asymmetrical?) should not be allowed to smoke;

[2] "Minor Maladies and Their Treatment", by L. Williams, M.D.; Sixth Edition; 1933. London: Baillière, Tindall and Cox; Australia: Angus and Robertson, Limited. Crown 8vo., pp. 423. Price: 10s. 6d. net.

wool and flannel next the skin are unhygienic. He weakens his arguments, however, by overstating his case. Many useful prescriptions are given which can be recommended as having stood the test of time and repeated trial. One which he designates "time honoured" contains ammonium carbonate and syrup of squill which are, of course, incompatible. The chapter by Dr. I. G. Cobb would in itself make the book worth buying. In it Dr. Cobb quotes Murray's treatment of chorea with fifteen drop doses of *liquor arsenicalis* thrice daily. He claims credit, however, for discovering that if necessary the treatment should be continued for a second week and that when arsenic fails, trional will often succeed. These two points are strongly emphasized by Murray.

Those who already own one of the earlier editions of the book would find the latest edition also worthy of a place on their book shelves.

THE CELLULAR ACTION OF DRUGS.

To all those interested in the application of scientific principles in therapeutics, Professor A. J. Clark's recently published lectures on "The Mode of Action of Drugs on Cells" will appeal as both an interesting and timely survey.[1]

Already in his "Applied Pharmacology" he had shown the influence of the application of critical experimental examination to an exposition of the subject, and the volume under review is no less stimulating. After outlining what is known of the cell as a physico-chemical system, the author proceeds to a consideration of the number of molecules of an active drug likely to be involved in affecting specific cellular processes, and it is no exaggeration to say that this chapter reads more like one of Sir James Jeans's interesting accounts of astrophysics than a chapter on the nature of drug fixation. Although in their nature rough approximations, the calculations provide a stimulating and entirely new viewpoint for the beginner in pharmacological research. The introduction of the "active patch" conception to explain the nature of fixation of certain drugs indicates the value of Chapter III, where, if anything, a more extended reference to the work of Quastel as it affects the relation of absorption to enzyme action, would have been useful.

Later references in this connexion to the consideration of the charcoal model by Rideal and Wright will prove instructive to those whose reading cannot cover so wide a field. The author's final conclusion on the application of physico-chemical laws to cell-drug reactions, "that any relations found are likely to be only first approximations", summarizes his views on the whole of his subject, and if they are depressing to those who find satisfaction in mathematical expressions, they are comforting to those whose views tend to comprehend the forest rather than to consider the individual trees. His conclusions from the work done on the kinetics of cell-drug actions support this view, for here the response to drug action may show a lag due to secondary processes, and the time measured is not the time taken for the initial drug action.

Perhaps the most interesting chapter is that dealing with variation in populations, either of cells or individuals. The work of Tattersfield and Gimingham, and of Percival, to quote only two examples given in this discussion of relation between drug concentration or degree of fixation and incidence of response, is most instructive, and the medical profession has everything to gain from the loss, here presaged, of the term idiosyncrasy from its vocabulary. This same variation is held to be responsible for the distortion of the exponential relation between drug concentration and drug fixation which results in the sigmoid concentration-action curves obtained. Throughout these discussions the author's refreshing mode of exposition of the historical development of conceptions, makes them interesting reading for the beginner. His conclusions no less so, for example: "This simplicity" (of the monomolecular and quantum theories of cell destruction) "is however in itself absurd, for it involves the assumption that the response of living cells is simpler than is that of relatively simple colloid systems".

The chapter on theories of drug actions is almost entertaining, as is also that on vitamins and hormones. A statement like the following (page 216) dealing with the number of factors involved in the extent of insulin hypoglycaemia indicates again the author's "biological" outlook: "This result agrees with the general principle that if there are considerable number of factors that vary independently, then the addition of one more may make very little difference". One feels acutely the absence of a mark of exclamation, and the concluding chapters on radiations and temperature effects complete an intensely interesting survey of a widely scattered literature on the subject of the action of drugs on cells. We owe a debt to Professor Clark for having in so small a compass reviewed so wide a field of investigation, the considered results of which make his book invaluable to the research worker in pharmacology. Is it too much to hope that it might form the beginning of a work in its own field like that into which Fraenkel's "*Arzneimittelsynthese*" has developed, and so become a unique standard work of reference, to which the author's quotation from William of Occam might well apply—"*Entia non sunt multiplicanda praeter necessitatem*".

[1] "The Mode of Action of Drugs on Cells", by A. J. Clark, B.A., M.D., F.R.C.P., F.R.S.; 1933. London: Edward Arnold and Company. Demy 8vo., pp. 305, with illustrations. Price: 18s. net.

THE CLASSIFICATION AND DIAGNOSIS OF HEART DISEASE.

A small volume comes from the Heart Committee of the New York Tuberculosis and Health Association; it has been adopted as the standard scheme of classification by the American Heart Association.[1] It is intended to improve upon and replace that issued in 1923. All physicians will agree that not only a common nomenclature but uniform criteria for classification is necessary from an international point of view for statistical and other research, apart from considerations in the passage of a patient from one physician to another, especially, for example, in Europe. The personnel of the Committee who have done their work so efficiently is a guarantee of its accuracy and completeness.

The whole scheme is clearly set out in sections under the titles, ætiological diagnosis, anatomical diagnosis, physiological diagnosis (which included the arrhythmias), functional capacity and provision for possible and potential heart disease.

Every possible contingency and combination encountered in clinical practice are covered in tables and each diagnosis is clearly defined and a list of the appropriate criteria is given. These lists have been purposely kept as short as possible to avoid any overlapping or confusion. If, for example, arteriosclerosis and hypertension occur together, they are put down separately. Diabetes would be placed under the title of "accompanying disease". Many labels familiar to English physicians are omitted, for example, atheroma, incompetence, bacterial endocarditis, occlusion *et cetera*.

The table for the differentiation and description of heart sounds and murmurs was urgently called for. Sounds are either normal, faint, loud or absent as regards intensity; normal, sharp, booming, split, reduplicated or ringing as regards quality; and normal, short or prolonged as regards duration. Murmurs are either blowing, harsh, musical, rumbling, crescendo or decrescendo. Teachers of clinical medicine or pathology and hospital authorities should, in the broadest interests of this subject, change and insist on the adoption of the scheme as a whole. Students must

[1] "Criteria for the Classification and Diagnosis of Heart Disease", by the Criteria Committee of the Heart Committee of the New York Tuberculosis and Health Association, Inc.; Third Edition; 1932. New York: New York Tuberculosis and Health Association. Demy 8vo., pp. 141, with illustrations.

inevitably learn all the alternatives of clinical jargon and other equivalents for these diagnoses from the multiplicity of their teachers, but they should, as far as is practicable, participate in a rational vocabulary such as this.

Appendices are added on radiological and electrocardiographic diagnosis to which the same arguments apply. Physicians concentrating upon cardiology should set the example and the little volume should be housed with their case histories and should be close to them when they are compiling their reports. The clear clinical descriptions and complete illustrations to the appendices give the work a text book value and make it worth reading even from this standpoint. It is to be hoped that other catalogues will follow concerning the remaining bodily systems.

MATERIA MEDICA AND THERAPEUTICS.

WHITLA'S text book is always welcome and the twelfth edition maintains the high standard of the previous issues.[1] All former editions were under the personal and sole supervision of Sir William Whitla, who can no longer apply himself to the task. However, Professor Gunn and his colleagues have ably carried on the work, which is thoroughly up to date. The arrangement of the book differs from that of most text books on *materia medica* in that the drugs are arranged alphabetically, which may be of advantage in some ways. Groups of therapeutic agents, such as alteratives, analgesics, oxytocics, vermicides and so forth are indicated in another part of the book. A special part is devoted to a list of the pharmacopœial preparations with descriptions and doses, being separated entirely from the therapeutics. Objections to this plan are appreciated by the authors, but the method will greatly facilitate the work of the student. The part devoted to pharmacy is excellent and is illustrated with appropriate figures. Part II dealing with prescription writing has timely observations on incompatibility and an extensive Latin vocabulary. Unfortunately the prescriptions throughout the work do not conform to the recommendations of the new pharmacopœia which discountenances the use of Roman numerals and symbols or the employment of drachms. The part concerned with therapeutics is excellent and a list of non-official remedies contains everything worth recording. Here we note several things of interest to Australia, such as our native leech, Duboisia and *Euphorbia pilulifera*, both plants being indigenous to Australia. The latter is often termed snake weed, not snake root as appears in the text. *Abrus precatorius* is also indigenous to tropical Australia and is known as jequirity. It is reassuring to learn that the barbarous treatment of trachoma by this preparation has been abandoned. The areca palm, yielding arecoline, also occurs naturally in Queensland and New Guinea. Of anti-pertussis vaccine the authors say that there is no certainty that it is of value either in treatment or prophylaxis. Antivenene also is stated to be specific only for the bite of the same genus of snake as that from which it was prepared. As a matter of fact it is specific only for the same species. No mention is made of Australian work on an antivenene. As regards gland substances and other tissues administered today in wholesale fashion the following is salutary:

> Unfortunately in spite of wide criticism, the unhealthy tendency persists to multiply preparations derived from various glands and tissues before knowledge allows that they have any claim to be considered as active physiological agents.

Again:

> Pancreatic extracts other than insulin and those made from testis and in many cases the ovaries and *corpus luteum* are inactive in the forms available or when given by mouth.

Errors are noted such as the dose of *Injectio Ferri* (page 129), given as fifteen to thirty mils, should read minims. *Emetinæ et Bismuthi Iodidum* should be *Emetinæ* (page 192). The new terminology is not meticulously followed. On page 32, *Acid. Arseniosi* should be *Arseni Trioxidi*. Similarly on page 163 *Arsenii Triiodidum* and *Arsenii Trioxidum* should be, in each case, *Arseni*. The spelling "carotine" is not in accord with recognised terminology, and, on page 178, the constitution of carbromal is not given accurately. No mention is made of the dangers of overdosage with thallium acetate nor the use of sodium thiosulphate in the treatment of arsenical or mercurial poisoning. With all these minor blemishes the book remains one of the best text books and an invaluable aid to students and practitioners alike.

[1] "Whitla's Pharmacy, Materia Medica and Therapeutics"; Twelfth Edition; Revised by J. A. Gunn, M.A., M.D., D.Sc., F.R.C.P. *et alii*; 1933. London: Baillière, Tindall and Cox. Crown 8vo., pp. 657, with illustrations. Price: 12s. 6d. net.

DERMATOLOGY AND ITS HISTORY.

DR. WILLIAM A. PUSEY, in providing us with "The History of Dermatology", has further increased the already heavy debt owed to him by the medical profession.[1]

For the first time a reliable history of dermatology is available in English. The book is beautifully got up, the paper, printing and illustrations are worthy of the highest praise. The volume consists of 273 pages, including a preface, introduction, nine chapters, an historical index of dermatology and a general index. The thirty-two illustrations show reproductions of various great dermatologists and facsimiles of the frontispieces of old text books of dermatology. We are skilfully taken through the ages of dermatology commencing at 3000 B.C.

It is only within the last one hundred and fifty years that dermatology has become a specialty and it is interesting to note that lepra did not connote leprosy till after the middle of the nineteenth century. Egyptian mummies studied by Elliot Smith did not show signs of leprosy.

Hippocrates was the first classiner of dermatoses, whilst Galen was the first recorded clinical dermatologist. The earliest description of smallpox and its differential diagnosis from measles was that of Rhazes, the Persian. During the middle ages the rapid increase in leprosy led to an intensive study of skin diseases and to the establishment of special hospitals for the care of the sufferers. The year 1500 saw the arrival of syphilis which led to a great increase in the attention paid to skin troubles.

Jean Astruc (1684-1766) is to be regarded as the founder of modern dermatology and not the better known Robert Willan, of almost a hundred years later. Willan's attempts at classification led to clear descriptions of dermatoses being given and cleared up much of the fog due to vague definitions. Willan was the first author to introduce the use of coloured plates in his text book.

The commencement of the nineteenth century saw the rise of the French school of dermatology which was brought about by the setting apart of the Paris St. Louis Hospital for the special care of skin diseases.

The preeminence of the French lasted till the middle of the century when the leadership passed to the Central Europeans headed by the great Hebra of the Allgemeine Krankenhaus of Vienna. In his first year in charge of the skin clinic Hebra found that nearly all his cases were scabies.

Every dermatologist should own this book and it will be read with interest by all who make a study of medical history.

[1] "The History of Dermatology", by W. A. Pusey, A.M., M.D., LL.D.; 1933. Springfield (Illinois): Charles C. Thomas; London: Baillière, Tindall and Cox. Demy 8vo., pp. 236, with illustrations. Price: $3.00 net.

The Medical Journal of Australia

SATURDAY, OCTOBER 7, 1933.

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PREVENTIVE DENTISTRY.

More has been written in recent times about dental sepsis as a cause of disease than about almost any subject. Well nigh every second pathological condition manifest in man has been attributed to infective foci in or around the teeth. In no other field has enthusiasm run riot to such an extent, and in witness of this enthusiasm we have an edentulous multitude, many of whom are unrelieved of their bodily ills. Even those who follow with cautious step the cult of focal sepsis and its eradication are sometimes forgetful that the formation of foci may be prevented. It is not so long since medical practitioners became alive to the possibilities of preventive medicine; not many have become alive to the possibilities of preventive dentistry. If enthusiasm for prevention were as great as the enthusiasm for cure by extraction of teeth, there would be fewer infective foci—medicine would be one cult the poorer and humanity would be by many teeth the richer.

There is no need, in a journal intended for medical practitioners, to discuss the importance of healthy teeth and of a clean mouth; every one recognizes that septic teeth and a dirty mouth may be the cause of disease in other parts of the body. But since prevention of dental sepsis, even if known to be desirable, is not always put into practice, the methods of prevention should be emphasized. The taking of food has long been looked on as likely to lead to a dirty mouth. When in olden days the prophet Amos wished to reprove the people of Israel for idolatry, he described the famine that had been visited upon them by saying that the Lord God had given them "cleanness of teeth" in all their cities. At the present day we know that food, particularly of certain types, if allowed to remain round the gingival margins, is likely to give rise to dental caries, unhealthy gums and so forth. It has remained for modern research workers to show that food of a certain vitamin content, when given to children, will endow them with teeth, healthy, hard and resistant to caries. The first steps in preventive dentistry should therefore be made in the earliest years of life. It is the duty of the medical practitioner to teach parents and guardians how the children in their care should be fed. As children are growing up they should be taught how to clean their teeth—an art not known to all adults—and when to clean them. As the years pass the children should be submitted to regular dental inspection; irregularities and malocclusion should be corrected at a suitable stage, and caries should be treated as soon as it makes its appearance. The regular inspection of teeth should be carried on right through life. These facts are well known, but are not always taught. It would be interesting to know how many medical practitioners submit themselves or have the members of their families submitted to regular dental inspection. That medical practitioners do not always practise as they preach is germane to the present discussion, for, if they did, they would become more insistent and more effective in their preaching.

While medical practitioners can, if they will, do a great deal on their own initiative to further the practice of preventive dentistry, they will be most effective if they cooperate with the members of the dental profession. In this issue we publish an article by Mr. E. Brangwin Green, the Chairman of the Dental Health Education Department of the Australian Dental Association. Mr. Green shows what his organization has done and is trying to

do in the dental health education of the people in New South Wales. He also enumerates several ways in which the members of the medical profession can further the work. We commend his article to the careful consideration of members of all the Branches of the British Medical Association in Australia—preventive dentistry is a necessity for all the States of the Commonwealth. There is no doubt that medical practitioners, once they have been seized with the importance of preventive dentistry, will be willing to cooperate with dental surgeons. To make such cooperation effective it is necessary that dental inspection and treatment shall be available to all members of the community. At present there are difficulties. There are very few free clinics for the inspection and treatment of persons who cannot afford to pay the full fees of dental practitioners. Moreover, inquiries in New South Wales have failed to reveal any arrangement for the dental treatment of so-called intermediate patients. No information is available regarding other States. This matter is one which needs to be dealt with as a matter of urgency. If the dental profession shows that it is in earnest, it will the more readily obtain the cooperation of the medical profession and the ideal of a *dens sanus in ore sano* will be nearer attainment.

Current Comment.

CARDIOVASCULAR SYPHILIS.

The importance of syphilis as a cause of vascular disease needs no stressing, for preeminently it is itself a vascular disease. The pathologist is able to demonstrate characteristic lesions in the blood vessels, and to point to the various end results that may be expected. Some of these results are easily distinguished in the living subject, for example, aortic valvular disease, though it may not always be simple to be sure that this particular anatomical change is due to the ravages of the *Treponema pallidum* and not to those of rheumatic infection. When the question of cardiac muscular damage comes up, however, its recognition is not so straightforward, owing to the impossibility of knowing the exact state of the coronary arteries during life, and also of the exact degree of pathological change that may have crept, as it were, almost unawares into the muscle fibres themselves.

E. Noble Chamberlain, in discussing this problem, begins by stating the criteria necessary for a diagnosis of syphilitic aortitis.[1] He remarks truly that early recognition of a lesion of the aorta is very important, for it may be possible to forestall further serious consequences by instituting early treatment. The nature of the sounds heard in the so-called aortic area are not conclusive. The ringing second cardiac sound, especially in persons over fifty years of age, even in the absence of arterial hypertension, is not certain proof. Neither is the presence of murmurs heard in this region. But when either of these signs is noted, when the patient suffers pain and breathlessness, and when there is perhaps some demonstrable dulness in the great vessel area, the diagnosis of aortitis is strongly suggested. Even then it is not certain that syphilis is the cause. To feel reasonably sure on this point it is necessary to have serological evidence, given by the Wassermann reaction (though this may be difficult or impossible to elicit, even after a provocative dose of arsenic), or other stigmata of a syphilitic infection, such as characteristic changes in the nervous system. Aortic dilatation is most probably due to syphilis, and it should always be looked for. In regard to those patients who show signs of a frank aortic regurgitation, the distinction between rheumatic and syphilitic disease has to be made, for syphilis and rheumatism are the most likely causes. Chamberlain remarks that subacute endocarditis must also be excluded; this is sound advice, for infection, that is, active infection, is not infrequently overlooked in the survey of a patient who has a cardiac valvular lesion. The history, though this is often misleading, the coincidence of mitral disease, the radiological evidence and the electrocardiographic findings, when taken together, will generally enable a correct opinion to be formed.

On the subject of syphilitic myocarditis there is less general agreement. Various authors are quoted in support of the frequency of syphilitic disease of the cardiac muscle, and others against it. Chamberlain sums up by saying that in spite of conflicting statements most workers admit that involvement of the myocardium is by no means rare. The symptoms likely to lead to suspicion that a patient is suffering from syphilitic myocarditis are in part those of other myocardial lesions, breathlessness, palpitation, pain in the precordial region, and nocturnal distress, and in part those suggesting other visceral forms of syphilis. In other words, there cannot be said to be an absolutely characteristic picture. Attacks of the so-called cardiac asthma, for example, occurring at night, are common, but by no means pathognomonic. Some workers have claimed a distinctive picture in the electrocardiogram, but, though the evidence yielded by this method strongly supports a diagnosis of cardiac, vascular or nutritional disturbance, it does not point to a definite ætiological factor.

It will be seen, therefore, that when the pathologists are not in entire agreement, the clinician is set a task that is apt to prove difficult. Perhaps the best way to put the matter is to point out that

[1] *The Lancet*, July 1, 1933.

in cardiac disease, particularly that centred in the main around the aorta and its valve, syphilis must always be thought of, it being remembered that one or two "negative" findings may not exclude it (for example, a failure to react to the Wassermann test). But it should also be remembered that even the known presence of a syphilitic infection, active or latent, does not preclude the possibility of other vascular disease, such as arteriosclerosis. The course of syphilitic cardiac disease is not a favourable one. Unfortunately there may be a comparatively long latent period, when symptoms are few or, indeed, are absent. It is during this quiet period that treatment is likely to modify the rather gloomy prognosis, and therefore an exact ætiological diagnosis is most to be desired. Bismuth and iodides are advised by Chamberlain, followed perhaps by the cautious administration of some organic arsenical preparation. In this he is in accord with accepted practice. Though it may be sometimes a matter of careful thought and judgement to direct treatment wisely, the more difficult task is to select the patients for such treatment.

CHANGES IN THE LIVER IN CATARRHAL JAUNDICE.

The so-called catarrhal jaundice is not an uncommon disease, but as it is fortunately not usually dangerous to life, there is little accurate *post mortem* information available concerning it. The similarity of onset and early signs and symptoms observed in this form of icterus and the more severe and lethal type known as acute or subacute yellow atrophy of the liver has often been noted. In fact it is a diagnostic point of some importance. More significant still, it has inspired the view held by some authorities that "catarrhal" jaundice, either epidemic or sporadic, is due essentially to the same process as yellow atrophy of the liver. If this be true, it is all the more important to secure accurate pathological data of both morbid states.

J. F. Gaskell has communicated a full account of the autopsy findings in a fatal case of epidemic catarrhal jaundice.[1] In his article he reviews the scanty records of such investigations, and points out that the usual findings have been an intense infiltration of round cells in the liver, chiefly round the portal canals or under the capsule, together with degeneration of the hepatic cells. The suggestion is made by one writer that the fatal case he describes was really one of acute yellow atrophy. But no recorded case in the literature throws any light upon the condition existing within one week of onset. This information is forthcoming in Gaskell's case. The patient was a child of five years who came to hospital for removal of tonsils and adenoids. She was apparently well, but subsequent investigation proved that in the village where she lived there was an epidemic of catarrhal jaundice at the time. All the other sufferers from this epidemic illness were but mildly affected and made uneventful recoveries. This little child was, however, apparently operated on on the eve of the manifestation of an attack of jaundice, for the following day she was jaundiced and febrile. Post-operative hæmorrhage was prolonged and troublesome, and recurred on the following day with great severity. In spite of the administration of calcium salts and whole blood, both by intramuscular injection and intravascular transfusion, the child died exsanguined.

Post mortem the liver was swollen and pale yellow. The gall-bladder contained bile, but the contents of all the bile ducts, both within and without the liver, were completely colourless. Microscopically the capsule of Glisson was found to be infiltrated with inflammatory cells, and the cells of the liver parenchyma were swollen and degenerate. Bile was not found either in the ducts, the liver cells or Küpffer cells, and fat also was absent. The larger bile ducts had intact epithelium and were not blocked. The inflammatory reaction was not confined to the perivascular or pericanalicular tissues, nor even especially grouped there, but was specially noted in the interlobular ramifications of the capsule. The primary change was considered to be in the liver cells themselves. In numbers of places freed and degenerate liver cells were seen in hepatic and portal veins, having apparently been expressed from the sinusoids. Thus the essential lesion was an acute hepatitis and there was no blocking of the bile ducts. No spirochætes could be found in the liver, which is in accordance with the results of other investigations in England and elsewhere. Apparently the common epidemic jaundice is quite distinct from spirochætal jaundice or Weil's disease.

The consideration of this case leads us to favour the hypothesis that subacute yellow atrophy may occur as a more severe manifestation of the common benign infectious catarrhal jaundice. Between 1925 and 1927 there was an extensive epidemic of infectious jaundice in Sweden, and during this period ninety-seven cases of subacute liver atrophy were recorded, the youngest patient in the latter series being aged fifteen years. Gaskell's case serves also to link up these two apparently distinct conditions, and he points out that a similar though more severe degree of cellular destruction might easily have produced the changes characteristic of acute atrophy. The chief cause of the patient's death was loss of blood. No doubt the existing liver damage predisposed to an hæmorrhagic state, and administration of an ether anæsthetic probably contributed somewhat also to the lowering of the hepatic reserve. A point of some interest concerns treatment. Biliary drainage with the duodenal tube is employed by some. Any good results must surely be due to the encouragement of duct drainage only and not to the removal of an alleged mucous or catarrhal obstruction. It would therefore seem reasonable to expect that equally good results could be obtained by methods simpler and more comfortable.

[1] *The Journal of Pathology and Bacteriology*, March, 1933.

Abstracts from Current Medical Literature.

MEDICINE.

Pernicious Anæmia.

P. J. Fouts and L. G. Zerfas (*The Journal of the American Medical Association*, July 15, 1933) recall that 300 grammes of whole liver (12 to 14 grammes of dried powder) or 200 to 300 grammes of fresh hog stomach tissue (30 to 40 grammes of desiccated material) by mouth will produce the maximal reticulocyte response in patients with pernicious anæmia in relapse. The potency of liver is increased after digestion in normal gastric juice, and Walden and Clowes obtained a very active preparation by the interaction of liver or liver extract with small amounts of fresh hog gastric tissue. This latter preparation was called "Extralin". Twelve capsules of this substance, of five grammes each, gave a response equal to 300 grammes of whole liver, and were said to be a satisfactory maintenance dose in pernicious anæmia. The response was slower than the response to liver extract, and as the hæmoglobin content of the blood did not reach normal, indicated the necessity of giving iron; 45 grains (three grammes) of iron and ammonium citrate daily were found effective.

The Hanganatziu Deicher Reaction in Angina with Mononucleosis.

U. Friedemann and P. Bier (*Deutsche Medizinische Wochenschrift*, March 24, 1933) write that Hanganatziu in 1924 found that the blood serum of people who had received serum for therapeutic purposes agglutinated very strongly the red blood cells of another species. Deicher discovered this reaction independently and found that it occurred seven to eight days after the injection, reached its maximum twelve to thirteen days later, and was present for months afterwards. The agglutination titre is often very high, even up to one to 250. This reaction was also found positive in nineteen cases of angina with mononucleosis and also helped to differentiate this disease from six cases of lymphatic leuchæmia.

The Auricular Rate in Auricular Fibrillation.

F. Scheltong and E. Sicks (*Münchener Medizinische Wochenschrift*, March 10, 1933) write that cases of auricular fibrillation occurring early in life are mostly tachycardia. In the course of months and years the high auricular rate decreases, even in untreated cases. The slow type is regarded as a result of functional damage of the bundle of His, and also due to damage of the cardiac muscle and circulation, owing to the rapid rate. An auricular rate below 70 is due to organic bundle damage and indicates a poorer condition of the heart. In contrast, the rapid rate indicates a good condition of the cardiac muscle and has a better prognosis. Prognosis becomes worse if cardiac failure ensues. With early and sufficient strophanthin treatment one can occasionally restore normal rhythm, and if this fails, quinidine should be used. If regularity of the pulse cannot be obtained, one must attempt to decrease the auricular rate.

Whooping Cough.

T. Madsen (*The Journal of the American Medical Association*, July 15, 1933) discusses the prophylaxis and treatment of whooping cough by vaccination. In Denmark the vaccine is made from several recently cultivated strains of Bordet-Gengou bacilli; its strength is 10,000 million per cubic centimetre. Three injections of 0·5, 0·7 and 1·0 cubic centimetre are given subcutaneously or intramuscularly at three or four day intervals. Two deaths have been reported in Denmark following vaccination of infants less than one month old; and though this is a rare event, children under one month old are not now vaccinated. In the Faroe Islands epidemics of pertussis occurred in 1923 and 1929. In all, 3,926 children were vaccinated, with six deaths; and 1,073 were not vaccinated, with 26 deaths. In the first epidemic there was little result in the prevention of the disease, but in the second epidemic 458 out of 1,832 vaccinated children did not contract the disease, whereas only eight out of 446 non-vaccinated escaped. These figures indicate considerable value both in prophylaxis and in mitigating the results of infection. The best results were obtained when vaccination was completed one week before the onset of the disease. In these epidemics the vaccine was made from young strains, the dose was rather large, a total of 22,000 million bacteria, and the vaccination was completed shortly before the onset of the epidemic.

Arterial Hypertension.

Plauquer, Riser and R. Sorel (*La Presse Médicale*, April 1, 1933) have reported researches on the pressure in the spinal fluid in patients with arterial hypertension. The arterial, venous and spinal fluid pressures were observed in twenty normal persons and in twenty who were subjects of hypertension. The venous pressure was estimated by the manometer of Claude (normal 11 centimetres of water). Those patients with arterial hypertension who also showed venous hypertension (14 to 29 centimetres of water) were found to have a high spinal fluid pressure; measured by Claude's manometer the normal pressure of the spinal fluid was 14 to 22 centimetres of water with the patient lying, and 30 to 42 centimetres with the patient sitting. It is probable that the increased pressure in the spinal fluid plays some part in the causation of the changes in the *fundus oculi* observed in subjects of arterial hypertension, such as venous congestion and œdema of the optic disk. In these patients, when the venous pressure is raised, the spinal fluid pressure is also increased; and treatment by purgation, venesection, digitalis, diuretics, or other measures of depletion may be expected to yield the best results.

Asthma and Pulmonary Tuberculosis.

W. E. Foggie (*Edinburgh Medical Journal*, March, 1933) discusses the relationship between asthma and pulmonary tuberculosis. He notes the contradictory statements found in the literature, some authors finding a very close association, others regarding the maladies as absolutely antagonistic. It is generally admitted that an asthmatic rarely contracts pulmonary tuberculosis, while it is even rarer to find a tuberculous patient suffering from asthma. Patients with chronic bronchitis may have asthmatic attacks; the signs of these diseases will mask all clinical evidence of a localized lesion, though it can be demonstrated radiologically, and tubercle bacilli will be found in the sputum. In a series of 373 patients suffering from bronchitis and asthma, 13 were found to have tuberculosis as well. The author notes the work of Harkavy and Hebald, who examined the sensitivity to foreign protein of 18 patients with both asthma and tuberculosis over a period of four years. Nine patients reacted to foreign proteins of the inhalant group, and all were freed of attacks by removal of the offending protein. The nine other patients all had respiratory sepsis, either sinusitis or bronchiectasis. In all these cases foreign proteins or complicating respiratory infection and not tuberculosis were the existing causes of the asthma. The author agrees that tuberculin is probably thus only of value in asthma as a foreign protein. He concludes that though there are associations between the two diseases, there is as yet no proof of a direct endogenous action of the tuberculous process in the production of true asthma.

Duodenal Ileus.

R. P. Rowlands (*The Lancet*, May 27, 1933) discusses the etiology, diagnosis and treatment of duodenal ileus. It is frequently associated with visceroptosis, especially where a mobile, pendulous and loaded right colon and caecum drag upon a short mesentery, the root of which compresses the duodenum against the lumbar spine. The author has obtained a cure in a case in which the cause was a congenital band compressing the second part of the duodenum just before it passes under the transverse colon. He has also removed a duodenal pouch arising from the posterior wall of the fourth part of the duodenum, which, when loaded, pressed upon the duodenum and caused duodenal dilatation with attacks of vomiting. Right lumbar nephrectomy has caused kinking of the third part of the duodenum, as have also adhesions following posterior gastro-jejunostomy. Other causes found were tuberculous and

carcinomatous glands. Indigestion, often dating from childhood, acute, subacute, or chronic attacks of painful distension in the epigastrium during and soon after meals, flatulence, nausea and anorexia are the commoner symptoms. The attacks generally last for two or three days, and are repeated at frequent and diminishing intervals. Sometimes vomiting gives relief. Generally the symptoms are relieved by lying down with elevation of the pelvis. During an attack the dilatation and stasis with reverse peristalsis may be demonstrated by radiographic examination. Clinically the condition has been mistaken for almost any of the commoner abdominal conditions, but particularly for gastric and duodenal ulcer. The medical treatment consists in adoption of the recumbent position with elevation of the pelvis and, if necessary, gastric lavage and duodenal aspiration. Postural treatment, abdominal exercises and supports may result in complete and permanent cure. Surgically, duodenojejunostomy is nearly always the best operation.

Idiopathic Exudative Pleurisy.

H. Schottmüller (*Münchener Medizinische Wochenschrift*, May 26, 1933) writes that idiopathic exudative pleurisy is only apparently a separate disease and its onset closely simulates croupous pneumonia. Clinical and X ray examination fail to reveal any changes in the lungs. Undoubtedly the cause is tuberculosis, although it is often difficult to prove this clinically. From this it follows that as a rule the condition is not due to a tuberculous pleurisy, but is the result of toxins arising from a tuberculous focus in the lung or mediastinum. This is the same association as that which occurs in the pleurisy of croupous pneumonia. As a contrast, *polyserositis tuberculosa* presents a different picture, but this disease can have a favourable course. Usually it is chronic and due to an anatomically recognizable tuberculosis of serous membranes. The commonest seasonal occurrence is in spring, and to a less degree in autumn. The commonest age of involvement is between twenty and twenty-five years, and the end result is usually satisfactory. Indeed, the slow resorption prolongs the convalescence, and this may take years. The treatment of the effusion varies with the clinician. The author always aspirates any effusion greater than a quarter of a litre, since he believes that by doing this the convalescence is shortened. If obvious destructive processes are present in the lung, which is very rare, then a gas replacement should be carried out. The duration of the rise in temperature lasted on an average twenty-one days in males and fourteen days in females. From then till the forty-second day subfebrile temperature occurred, and at this time the patients were free of fever and could get up. Should an exudate form after the first paracentesis, it should be removed and the aspiration should be repeated if necessary. All of the exudate should be removed. In the author's experience, the duration of the illness is shortened, for the patients can get up on the forty-second day. Conservative therapy takes much longer, and von Gsell states that on an average these patients need ninety days. The end result of this therapy was quite as good as that of conservative therapy.

Endothoracic Goitre.

P. Clairmont (*Münchener Medizinische Wochenschrift*, April 28, 1933) writes that the diagnosis of endothoracic goitre depends on indirect symptoms, namely, pressure on surrounding organs resulting in a turgid face, cyanosis, widened veins in the neck and anterior thoracic wall, laboured, painful breathing, with the assistance of the accessory muscles, inspiratory and expiratory stridor, a widened, fixed thorax, generalized rhonchi, and the slow increase of respiratory difficulty with sudden increase of attacks of suffocation at night. The only proof is feeling an increased resistance behind the *manubrium sterni*, which is dome-shaped and non-pulsating and which moves with deglutition (if the goitre is attached to the trachea). X rays are most important in the diagnosis. The author nearly always finds an enlarged heart with this goitre as well as other pressure symptoms. If dysphagia occurs, it may either mean that the thyreoid is enlarging posteriorly or is causing the pressure by malignant infiltration of the œsophagus. In operations for this condition the sternum has rarely to be split, for usually the goitre can be displaced into the wound gradually by tension sutures. This technique avoids the risk of air aspiration, bleeding, damage to the pleura and sudden cessation of heart action and respiration.

The Ætiology of Pernicious Anæmia and Related Macrocytic Anæmias.

W. B. Castle (*Annals of Internal Medicine*, July, 1933) states the conclusions as to the ætiology of pernicious anæmia and other macrocytic anæmias arrived at as a result of the work of himself and other investigators during the last five years. Addisonian pernicious anæmia is a deficiency disease conditioned by the lack of a specific intrinsic factor present in normal human gastric juice. This intrinsic factor is a heat-labile substance not corresponding in its properties to hydrochloric acid, pepsin, rennin, or lipase and its function is to interact with an extrinsic factor in the food to produce specific hæmatopoietic effects (demonstrable in pernicious anæmia). The characteristics and distribution of the extrinsic factor correspond to those of vitamin B_2. Failure of the reaction between the two factors or non-absorption of the product may be expected to lead to the development of pernicious anæmia. The demonstration of the hæmatopoietic activity of various substances, such as arsenic, liver, kidney, brain, and even stomach preparations or the injection of milk or gastric juice, does not necessarily establish their ætiological relationship to the disease, despite their efficiency in its treatment. There are three possible mechanisms by the action of one or more of which pernicious anæmia may be produced. There may be a lack of the intrinsic factor, a lack of the extrinsic factor, or a failure of absorption or utilization of the product of their interaction. All cases of classical Addisonian pernicious anæmia in relapse so far studied have been mainly due to lack of the intrinsic factor. Sprue with macrocytic anæmia, however, has been successfully treated with diets rich in vitamin B_2, and so also tropical macrocytic anæmia and the macrocytic anæmia of cœliac disease. In certain cases of macrocytic disease the third possibility, defects of absorption, may be involved.

The Measurement of Proteinuria.

F. H. Lashmet and L. H. Newburgh (*The Journal of the American Medical Association*, April 29, 1933) describe a method of estimating the amount of protein present in urine, the specific gravity of which is being determined as a measure of the renal function. The specific gravity of urine is increased 0·003 by the presence of 1% dissolved protein. Any proteinuria below 0·3% need not be considered in the correction of the specific gravity. The method consists in the comparison of the turbidity produced by the addition to the urine of thiosalicylic acid with a stock solution made permanently turbid by the suspension of inorganic substances which are easily procurable. The stock solution is made by adding 50 cubic centimetres of 0·1 normal sodium hydroxide solution and eight grammes of copper sulphate (hydrous) to about 200 cubic centimetres of distilled water in a half litre volumetric flask and making up the 500 cubic centimetres with distilled water. After shaking the stock solution vigorously to insure uniform suspension, two cubic centimetres are transferred to a test tube (16 millimetres in diameter) and 23 cubic centimetres of distilled water are added. The turbidity of this mixture is identical with that produced by a 0·1% solution of protein made turbid by the addition of thiosalicylic acid. One cubic centimetre of filtered urine is placed in a test tube of the same diameter and 24 cubic centimetres of 2% solution of thiosalicylic acid are added. Comparison of this tube with the standard is made not sooner than three minutes or later than ten minutes after the thiosalicylic acid has been added. If the turbidity is the same as that of the standard, the urine contains 0·1% of protein. If the turbidity is greater, the urine must be diluted and the procedure repeated until a "match" is obtained. If the urine is diluted one in ten before it matches the standard, then it contains 1% of protein.

British Medical Association News.

SCIENTIFIC.

A meeting of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, East Melbourne, on July 5, 1933, Dr. W. G. D. Upjohn, the President, in the chair.

The Drainage Tube in Peritonitis.

Dr. A. J. Trinca read a paper entitled: "The Abuse of the Drainage Tube in the Treatment of Peritonitis, with an Analysis of 224 Cases of Diffuse Peritonitis Treated Without Drainage" (see page 465).

Dr. Basil Kilvington thanked Dr. Trinca for introducing a very important subject in an interesting manner. He said that there was much difference of opinion as to drainage in peritonitis, and everyone agreed that the prolonged and multiple drains of twenty years ago were a mistake.

The peritoneum had a marked resistance to infection, much more so than the lining of a joint, so that it would withstand many organisms. Probably no abdominal operation had ever been truly aseptic. The resistance of the peritoneum depended on the intact endothelium, which became phagocytic on occasions, and the cells migrated. The moral of this was when operating to damage this membrane as little as possible by vigorous handling or rough swabbing, or even by antiseptics introduced; so this last had been abandoned.

The second line of resistance was the white cells of the blood. When the peritoneal lining was destroyed, such as occurred in a definite abscess, the resistance was lost and everyone agreed to use drainage. The disadvantages of draining were incisional hernia, intestinal adhesions and possible fæcal fistula from pressure of the tube. One of the commonest instances of peritoneal soiling occurred when a pyloric ulcer gave way. Here a variable quantity of irritating fluid leaked into the peritoneum, but it was highly acid and usually comparatively sterile, though this last was not constant. Most of these patients recovered if the rupture was sutured and the fluid was removed, preferably with gentle use of a Sprengel pump and without drainage, provided the operation was done within twelve hours of rupture. Sometimes trouble occurred and the risk of this increased with delay in operating. Dr. Kilvington considered that the proper treatment was to use drainage in every one of these cases, however early the operation was done, by making a stab wound above the pubes. An occasional exception might be made where the stomach was empty at the time of rupture and scarcely any fluid escaped. The drain was left in no longer than forty-eight hours, for after this the peritoneum was shut off and the tube was walled off, leading only to adhesions.

Dr. Kilvington had never seen an incisional hernia after a stab wound, and in a few cases when the abdomen had been opened later on there were usually no adhesions present at all. Thus the objections to drainage were absent. Not infrequently in an undrained case there developed a collection in the pelvis, which gradually became infected from the bowel and required a second operation. Appendicitis furnished the great difficulty in the question of drainage. No one advocated using drainage when an inflamed appendix was associated with a certain amount of turbid, odourless fluid, which Dudgeon and Sargeant many years ago showed was actively bactericidal in nature. Neither did any surgeon hesitate to use a tube in a localized abscess where the peritoneal endothelium had been destroyed, even when the appendix itself had been removed.

The cases in doubt were those in which a gangrenous appendix had perforated and there was a spreading peritonitis associated with foul-smelling fluid (though the smell might be absent in streptococcal infections).

Dr. Kilvington's own practice was to remove as much fluid as possible by gentle suction with a Sprengel pump and to drain for thirty-six hours with a tube placed in the bottom of the pelvis through a stab wound. He stated that he regarded as of equal importance in very bad peritoneal infections an ileostomy to drain the paralysed bowel of its toxic products and the administration of the serum against the *Bacillus welchii*.

This class of case gave much anxiety to the practised operator and much more so to the young surgeon. No description from books would enable one to decide in an individual case, and Dr. Kilvington strongly urged the rule: "When in doubt, drain." A beginner would probably drain more frequently than an older man, but if a patient required drainage he needed it badly, and its omission might kill him.

Dr. Kilvington said that he would prefer to remain alive with perhaps an incisional hernia and some adhesions than to enter the next world with an intact abdominal wall.

Dr. Gordon Shaw said that it was a good thing to review surgical practice in any particular respect from time to time, and Dr. Trinca had done good service in making them consider whether the routine use of the drainage tube in peritonitis was sound practice, and in making those surgeons who used the drainage tube defend their position. Dr. Trinca had shown an extraordinarily good series of cases of peritonitis treated without drainage, on which he was to be congratulated; and he had made a very strong case for the abolition of the drainage tube.

However, Dr. Shaw was not convinced. It was interesting to look back some thirty years, when peritonitis was a very fatal disease, and to observe the change in the results of treatment which came about on the introduction of Murphy's principle of operating, namely, rapid opening of the abdomen, removal of the cause, if possible, and the placing of a drainage tube, the continuous use of saline solution, and the Fowler position.

They now had to ask themselves were these methods, which revolutionized treatment thirty years ago, unnecessary.

Dr. Trinca said that the peritoneum was capable of dealing with infection. Dr. Shaw agreed with him that it could do so up to a point, but asked was it wise to extend the powers of the peritoneum to their utmost, and was it not better surgery to assist the peritoneum in combating infection.

Dr. Shaw divided peritonitis into two classes: (1) Potential peritonitis, when there was non-infected or very slightly infected fluid in the peritoneal cavity, such as extravasated gastric or duodenal contents from a ruptured ulcer, or healthy bile, or urine from a healthy bladder. In such cases it was sufficient to evacuate the fluid, though if this could not be done completely, the insertion of a drainage tube into the pelvis for twenty-four hours was a safe and proper precaution. Such fluid became infected after a short time, and Dr. Shaw could see no useful purpose that it served if it was left in the abdomen as advised.

Another type of potential peritonitis was that present shortly after a perforation of an appendix. In this case there was a pool of sero-pus surrounding the appendix, the dissemination of which was prevented or delayed by the sticking together of adjacent coils of intestine and omentum. In this case there was no doubt that all that was necessary to be done was to remove the appendix, and if the abdomen was closed without drainage, the peritoneum could deal with any infection which might have taken place.

The second type of peritonitis was what one might call actual peritonitis. In this case the effusion was infected with virulent organisms, and here Dr. Shaw stated that he regarded drainage as essential. It relieved intra-abdominal pressure and so diminished absorption of toxins, and it established a flow of fluid from the tissues to the exterior which washed out the infected material. Dr. Shaw said that it was impossible to tell whether the peritoneal effusion was infected or not, and moreover that the degree of infection varied in different parts. Dr. Shaw had never seen judicious drainage cause any harm. It did not increase appreciably the risk of post-operative hernia, as this was due to infection of the wound, and in any case the tube could be brought out through a stab incision. It did not cause fistula, this condition being due to giving away of sutures at the base of the appendix, and in such a case the track formed by the tube was

essential. Dr. Shaw remarked on the small number of cases of wound infection in Dr. Trinca's series and said that this must be attributed in part at least to the absence of the drainage tube.

Dr. A. E. Coates stated that he was impressed by Dr. Trinca's figures, and was "almost persuaded" to adopt his methods. On closer inspection, however, it was apparent that the figures referred to many cases which were not generally classified as peritonitis. One hundred and sixty-four cases were simple acute appendicitis of one day's duration. The general opinion was that these cases did not need drainage. The diagnosis of general peritonitis based on the appearance of a small amount of milky fluid appearing about the appendix when the abdomen was opened, was not justifiable. Dr. Coates regarded these cases as simple local effusion, and treated them without drainage. In cases of perforated appendix and gangrene of the caecum of mesenteriolum, a drain was necessary.

Dr. Trinca had maintained that by using a light anæsthesia he was able to locate the fæcoliths or other matter which had escaped from the bowel, and could be quite certain of clearing it out. This did not seem to be the experience of many surgeons who found the coils of bowel often distended, and the complete removal of all necrotic material was a matter of conjecture. A drain tube seemed th best means of playing for safety in these cases.

Dr. Coates did not agree that packing the bowel off was a harmful procedure. He much preferred to place a small pack or two in the wound, pushing the coils of small intestine away and protecting them before manipulating the acutely inflamed appendix. It might be on the point of perforation, or there might be a small abscess around it. The contamination of the rest of the peritoneal cavity with this material could hardly be regarded as of no importance. In cases of perforated peptic ulcer drainage was advisable in the pelvis by a suprapubic stab wound and also a tube in Morrison's pouch. In two cases in which Dr. Coates had followed the teaching of the non-drainage school, a second operation was necessary to evacuate pelvic collections and to relieve obstruction. Both patients died. The drain often discharged a large quantity of fluid in the first twenty-four hours. It could then be shortened if drainage subsided, and gradually removed in four to five days. The subphrenic space should always be examined in perforated ulcer. Often a large quantity of gastric contents would be found there, whereas little was seen about the site of perforation. It seemed wise to place a tube in the wound for a day or two, in case any food material or *débris* had been overlooked. Fleming had shown that 93% of cases of twelve hours' perforation gave a positive culture from the peritoneal fluid. Dr. Coates could not be sure of removing all this fluid and therefore appreciated the assistance of the drainage tube.

The problem was a difficult one. He (Dr. Coates) drained fewer cases than he had done formerly, but it was a difficult matter to instruct resident medical officers at hospital and students simply on theoretical grounds. They had to see the cases and learn by experience, as all surgeons did, which cases might be safely left and which required a drain. Until they had had that experience the advice, "when in doubt, drain", seemed the soundest.

Dr. Newton, in a recent series of over one thousand cases of appendicitis at the Melbourne Hospital, had drawn attention to the large incidence of residual abscess and other catastrophes following the appearance of St. Leger Brockman's article advocating non-drainage. Dr. Coates had opened four cases of residual abscess at that time, where resident medical officers had followed the new fashion.

The disadvantages of the drainage tube were exaggerated. In a series of 234 cases of intestinal obstruction at the Melbourne Hospital which were analysed recently, a large number of cases of obstruction by adhesion were found to be the result of previous attacks of appendicitis which had not been followed by operation. The adhesions obviously were the product of the plastic exudate and not due to the drainage tube. One only needed to visualize a piece of chewing gum gluing coils of bowel together and later stretching, to have a picture of how these adhesions produce obstruction.

Dr. Coates did not agree with Dr. Trinca that ileus was of sudden onset. It came on insidiously. He (Dr. Coates) did not like a silent abdomen. The patient who was completely free from peristaltic pain and could sit up and read the paper the day after the removal of a dirty appendix, often gave serious trouble. One felt happier if the patient had a few pains as the gas passed the ileo-caecal valve. A gentle laxative, such as cascara or calomel, after forty-eight hours, and an enema, if necessary, relieved the patient. The rectal tube was a valuable aid in helping in the removal of flatus. It was not necessary to obtain a large evacuation of the bowels, but as long as flatus was passed, there was no risk of ileus. The silent abdomen was ominous. If distension were marked, a hot foment to the abdomen and a rectal tube usually relieved it. Morphine was used freely in patients with large effusions of pus and a general peritonitis with little distension. When, however, distension was present at operation for the removal of the perforated appendix, in association with a widespread peritonitis, then not only a pelvic drainage tube, but also, in old or enfeebled patients, an ileostomy tube was introduced as well. If vomiting were persistent, the stomach tube was of great assistance. Rather than abolish the use of drainage tubes, there was much evidence to support the advocacy of more and better drainage tubes, namely, stomach tube occasionally where necessary, the judicious use of the peritoneal drain, the occasional use of ileostomy or caecostomy and the frequent use of the rectal tube.

Rather than depart from the methods of the older school and the traditions of the elders, as represented by Dr. Kilvington and Dr. Gordon Shaw, it would be better for the younger school to profit by their experience and feel their way in the use of the drainage tube.

Dr. H. Douglas Stephens referred to the use of the drainage tube in abdominal surgery in children. He said that Dr. Trinca's paper opened up an aspect of treatment in adults which contrasted strongly with his own (Dr. Stephens's) experience in children. The resistance to purulent infection of the peritoneal cavity in young children was very poor, possibly due to the lack of development of the omentum, and, as far as his own practice was concerned, he agreed with the views expressed by Dr. Coates. Two years ago he had, in accordance with modern tendency, tried to dispense with drainage in some cases of mild local infection of the peritoneum, but had gone back to the principle of "when in doubt, drain". He avoided purgation, but encouraged bowel action on the third day by using a rectal tube or enema or, better still, by inserting a gloved finger into the rectum. A small dose of morphine was advantageous for patients experiencing much pain, discomfort or restlessness after operation, and the application of a large abdominal compress also afforded much comfort. Children liked fruit juice drinks and took them as a rule readily after operation with relief to thirst and vomiting.

Dr. R. C. Brown said that the difficulty in twisting the intraperitoneal drainage tube in twenty-four hours was due to peritoneal lymph, produced by irritation by the tube, protruding into the small holes in the tube. For the same reason the coils surrounding the tube were glued together, effectually preventing the functioning of the tube. He rarely used an intraabdominal tube during his latter years at the Alfred Hospital, except in the case of abscess.

In ruptured duodenal ulcer he did not drain the pelvis and only rarely placed a very temporary drain in the left loin. In his experience it was very difficult and dangerous both to locate and evacuate the residual abscess, owing to the extrusive adhesions and friability of the surrounding bowel. It was better to allow the abscess to evacuate spontaneously into the bowel.

In abdominal inflammation he avoided purgatives altogether; he used morphine in one-eighth grain doses, repeated as required, and avoided worrying the patient with rectal injections of saline solution when once the patient could retain fluid by the mouth.

Dr. F. F. D'Arcy said that in Continental clinics it was usually possible to get a pathological report on the contents of a ruptured appendix or peritoneal abscess during the course of the operation.

Dr. John Kennedy congratulated Dr. Trinca on his original and stimulating address, but he himself did not belong to the school of "no drainage". However, he had to admit that in cases of peritonitis, in which he had used a drainage tube, he was struck by the extraordinarily small quantity of fluid drained by the tube, when compared with the large amount of fluid within the abdomen. A small quantity escaped during the first few hours, and then apparently nothing. He considered that drainage tubes did little more than make a track from the septic focus to the surface. In perforated gastric ulcer with gross extravasation of the stomach contents he invariably placed a tube into the pelvis, from which there was, as a rule, very free drainage.

Dr. Hugh Trumble said that he had started his surgical practice as an exponent of the use of the drainage tube and had found that as a rule very little pus came through the tube. Later, he heard the details of Dr. Trinca's methods and cautiously he modified his own practice somewhat along the same lines. He still used drainage in a greater proportion of cases than did Dr. Trinca, but he was using it less and less, and so far he had not had any reason to regret its non-use. As regards the use of morphine in acute abdominal inflammation, one and a half to two grains of morphine injected hyperdermically into dogs usually made them vomit and pass fæces, indicating an increased peristaltic action. It was not always sound to argue from animals to man, but he did not think morphine in small doses in man greatly inhibited peristalsis.

As regards the drainage of pelvic abscesses, he thought that these should, as a rule, be drained *per rectum* or *per vaginam*, as advocated by Barnard. This method was preferable to suprapubic drainage and did not appear to have attained the popularity it merited.

Dr. Trinca, in reply, said that as the subject he had introduced for discussion contained much controversial matter, he had expected and received a good deal of opposition to his views. He was disappointed that his arguments, based on pathological considerations, had not been appreciated, and he felt that various speakers had not based their opinions on pathological facts, but were still obsessed with the imaginary virulence of peritoneal exudates.

One of the big points he wished to stress was that the mortality from appendicitis was still too high. He felt that it would remain high as long as surgeons failed to appreciate the pathological aspects of peritonitis and continued the unnecessary use of the drainage tube. The pathological facts he had mentioned had been proved by experiment and he hoped the references he had quoted would be studied by his critics. He could not understand the difference in observation between other speakers and himself with regard to the amount of fluid which escaped through a drainage tube in the forty-eight hours following its introduction. He definitely disagreed with those who said that pints of fluid escaped from the tube. His (Dr. Trinca's) observation would suggest that little or none escaped until the tube was removed. The admission by Dr. Shaw, who first of all stated that he removed all the exudate and then inserted a tube, through which poured copious quantities of fluid, was a statement of two irreconcilable facts and an inconsistent argument. Dr. Shaw had said that after removing the exudate a further quantity formed rich in antibodies. He (Dr. Trinca) would like to know where these antibodies came from or, since the infection had been removed, what promoted their formation. Surely this was an unscientific argument. Also if, as Dr. Shaw stated, there was a preperitonitic stage when fluid was sterile, why remove it?

Dr. Kilvington had stated that the tube was not sealed until forty-eight hours after insertion. He (Dr. Trinca) could not accept this statement. In his opinion it was sealed very soon after insertion by the application of a mobile coil of bowel containing gas under atmospheric pressure; otherwise there would be a vacuum present after the abdominal wound had been closed. He could confirm the statement of Dr. R. C. Brown that after twenty-four hours it was very difficult to move the tube. It had been gripped tightly by the swelling of the tissues which surrounded it.

The list of cases analysed comprised all the cases of peritonitis which he had not drained. It did not represent the peritonitis cases which he had seen during the same period that had been drained for reasons already stated. His paper was not on the subject of drainage in peritonitis, but on non-drainage in suitable cases.

He was very disappointed to hear some of the speakers advocate the use of purgatives, and he could not understand their rationale. He particularly disliked calomel, a definite irritant poison, and he disagreed with Dr. Shaw, who had stated that calomel was particularly of use for patients who had nausea. In Dr. Trinca's opinion calomel would produce nausea and was one of the worst purgatives to use.

With regard to the harm that might be produced by the drainage tube, he had distinctly stated that the amount of harm would vary directly with the length of time the tube was left in. Many who used drain tubes left them in a long time. He had recently heard of a case in which a tube was sewn in with chromicised catgut and left in for nine days.

With regard to the statement that the cases illustrated were not cases of peritonitis and that he would not have classed them as peritonitis, Dr. Trinca claimed that as a pathologist as well as a surgeon his opinion on inflammation of the peritoneal cavity was at least as good as that of the surgeon. He would repeat that no case was included in the list unless enough exudate was present to escape when the peritoneum was opened. He had not included cases with an exudate in the bottom of the pelvis. The accumulation was at least as high as the anterior superior spine and was at least cloudy, in many of the cases frank pus being present.

In answer to Dr. Stephens, Dr. Trinca stated that with regard to peritonitis in children he had not had a large experience, but the results obtained in the treatment of those were sufficient to make him apply exactly the same principles of treatment to children as regards non-drainage and removal of the exudate.

Dr. D'Arcy had asked for bacteriological investigation of these cases. Dr. Trinca would produce a summary of six of the last cases he had treated where bacteriological investigation of the general exudate had been carried out.

With regard to the question of fæcal fistula, he did not think that this was due to the breaking down of a gangrenous caecum so much as to damage by a tube that was left in too long. He had occasionally seen cases in which the caecum was œdematous or suggestive of early gangrene, and if he was suspicious, had inserted a light gauze or rubber dam drain to this point for thirty-six hours.

Dr. Trinca congratulated Dr. Kennedy on his frankness in admitting that the results obtained in cases of peritonitis that he had treated with drainage were not so good as cases he had seen treated without drainage. In reply to Dr. Kennedy's question on the treatment that Dr. Trinca would adopt in the case of involvement of the caecal wall, Dr. Trinca stated that if he was in doubt of the condition of the caecum he would insert a rubber dam drain down to the doubtful spot.

In reply to Dr. Trumble, Dr. Trinca stated that he was very pleased to hear of the abolition of the drainage tube in Dr. Trumble's practice, and he was quite sure that if Dr. Trumble were to produce his figures his mortality in peritonitis would be very low. He was very interested to hear of the effect of morphine as a stimulant of intestinal peristalsis in dogs.

NOMINATIONS AND ELECTIONS.

The undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Anderson, John Ivor Harold, M.B., B.S., 1931 (Univ. Sydney), Coast Hospital, Little Bay.

Medical Societies.

THE MELBOURNE PÆDIATRIC SOCIETY.

A meeting of the Melbourne Pædiatric Society was held at the Children's Hospital, Carlton, on July 12, 1933, Dr. F. Kingsley Norris, the President, in the chair. The meeting took the form of a series of clinical demonstrations.

Bronchial Fistula.

Dr. H. Douglas Stephens showed a girl of three and a half years who had been admitted to the Children's Hospital at the age of eleven months with a left basal pneumonia. Following this she developed an interlobar empyema. A rib had been resected in the posterior axillary line and the empyema drained. The drainage, however, persisted continuously for two years until Dr. Stephens saw her for the first time in September, 1932. Thinking that the condition might be a chronic empyema, he opened up the cavity to see whether there was possibly a foreign body present or whether the cavity had become epithelialized, but he found instead that there was a direct communication with a bronchus.

The cavity was swabbed with gentian violet and packed, but no improvement followed. Two months later, having seen the article of Pool and Garlock in the *Annals of Surgery* in August, 1929, where it was advised to turn living muscle into the cavity, Dr. Stephens again operated and turned the muscle *pectoralis minor* into the cavity and stitched it in position. The wound had healed by first intention and the child completely recovered.

Twisted Ovarian Cyst.

Dr. C. H. Osborn showed a girl of four years and five months who had been admitted to hospital because of anorexia, vomiting and abdominal pain of four days' duration. On examination she had a temperature of 37·3° C. (99·2° F.), a pulse rate of 140, a thickly coated tongue, and tenderness and rigidity in the right iliac fossa. The pre-operative diagnosis was acute appendicitis, but at operation the appendix was normal. There was, however, a tumour in the pelvis, which was found to be a twisted ovarian cyst, and was removed. It was a teratoma containing smegma, hairs and calcareous material. Ten days later she developed intestinal obstruction through the ileum kinking around an adhesion and again had to be operated on. Following this she made an uneventful recovery.

Dr. Osborn showed the patient because of the great rarity of twisted ovarian cysts in children. Wakeley, in *Surgery, Gynecology and Obstetrics*, March, 1933, said that of all tumours of the ovary, 7% were dermoids or teratomatous cysts. They were very rare before puberty, the total number to the present time being under one hundred. In the past twenty-five years there had been no cases at Great Ormond Street, and only one at King's College Hospital. Dr. Osborn could find no record of a twisted ovarian cyst in childhood.

Carcinoma of the Sigmoid Colon.

Dr. Osborn next showed a girl of ten years who had been admitted to hospital with a story of vague abdominal discomfort for the past six months. Twice during this time she had had attacks of actual pain accompanied by blood and mucus in the stools and some vomiting. On admission to hospital she had some generalized abdominal pain, a temperature of 37·8° C. (100° F.), and a tender, rounded, and resonant tumour in the hypogastrium. Rectal examination showed that although the tumour was just palpable, it did not extend to the pouch of Douglas.

At operation the abdomen was opened by a mid-line incision and the tumour was delivered. It was surrounded by omentum. The abdominal cavity was packed off and an attempt made to unravel the tumour. During this the tumour broke and some pus escaped. It was determined with difficulty that the tumour was situated at the loop of the sigmoid. It was thought to be tuberculous. There was apparently no involvement of the glands. The tumour and a wedge-shaped area of mesentery were removed and a double-barrelled colostomy was performed. Recently, the spur had been divided and the colostomy was slowly closing. Section of the tumour showed it to be an adeno-columnar carcinoma with early colloid degeneration.

Dr. Osborn remarked that this condition also was fairly rare, although Phifer, in the *Annals of Surgery* in June, 1923, had reviewed 23 cases of carcinoma of the rectum and sigmoid in children under sixteen years of age. Mummery, Drinkwater and Ewing since then had also quoted such cases.

Tetanus.

Dr. R. L. Forsyth showed a girl of eleven years who had run a splinter into the left leg, above the knee, six days before admission to hospital. Twelve hours before admission she had developed stiffness of the jaw, the back and the left leg. On examination she had a temperature of 37·2° C. (99° F.) and a pulse rate of 96. There was some slight neck stiffness and trismus with rigidity of the left leg and the abdominal muscles. The condition was diagnosed as tetanus and treatment was carried out as follows. Daily, so long as any evidence of toxæmia remained, she was given anti-tetanus serum by the various routes of administration in the following total dosage: Intracisternally, 140,000 units; intravenously, 310,000 units; and intramuscularly, 150,000 units. Various sedatives were used also, but chiefly chloral hydrate by mouth and paraldehyde by the rectum. "Sodium amytal" was also used on two occasions. The wound was excised. After thirty-two days', during which time the patient's condition fluctuated considerably, she made a complete recovery.

Dr. Forsyth said that the main problems in the treatment of tetanus were the method of administration of serum, the amount of serum to be given, and the duration of treatment. This patient had been given 600,000 units, and although he would not have liked to discontinue the serum, he felt that much of it had probably been wasted. Much of the experimental work was contradictory, and investigations at other hospitals were not of much help. The case demonstrated that at least large amounts of serum given intrathecally did no harm, and also that so long as there were any signs of activity, they must continue with the serum. His impression was that after the initial flooding of the theca and blood stream with serum, they could carry on with much smaller amounts.

Dr. M. O. Kent Hughes said that from a review of the cases at the Children's Hospital, which he hoped later to publish, the following points seemed to stand out. The rapidity of onset of the symptoms seemed a more reliable prognostic guide than the length of the incubation period. Moderate dosage seemed to be just as effective as very large dosage. If the serum had tricresol added as a preservative, it was dangerous to use it intrathecally, but not so intravenously. One case, in which no local treatment was undertaken and in which recurrence took place three times in twelve months, seemed to indicate that excision of the wound was essential.

Macrocytic Anæmia.

Dr. T. Frank showed a boy of thirteen years with a history that for the past nine months he had had recurrent sore throats with enlargement of the cervical lymph glands. For the past three months he had become progressively paler and more languid. Two weeks previously he had had his tonsils and adenoids removed and the glands had decreased in size, but the pallor and tiredness persisted. There was nothing relevant in either his past history or his family history.

Examination revealed a well developed boy with a sallow, lemon-yellow complexion and an icteric tinge of the conjunctivæ. The spleen was not palpable and, apart from the cervical glands, no other glands were palpable. Examination of the blood showed that there were 1,680,000 red corpuscles and 12,600 white corpuscles per cubic milli-

metre; the hæmoglobin value was 60% and the colour index 2·0. The film revealed a general macrocytosis, some of the cells being very large. There were also large numbers of nucleated forms, particularly megaloblasts.

A fractional test meal revealed the presence of free hydrochloric acid in the gastric contents. The Wassermann, von Pirquet and Fouchet tests all failed to yield a reaction. Sufficient blood could not be obtained for a corpuscle fragility test. Bogomolov's test for urobilin in the urine gave a positive result. The boy was given a good mixed diet, cod liver oil emulsion 12 mils (three drachms) twice a day, iron and ammonium citrate 6·0 grammes (ninety grains) daily, and 0·22 kilogram (half a pound) of liver daily. Despite this intensive treatment the response was very slow, because in five months the red cells increased only to 2,570,000 per cubic millimetre. Also there was very little reticulocytosis. The hæmoglobin was now 44% and the colour index 0·86. The macrocytosis had disappeared also.

Dr. Frank said that although the findings on blood examination were like those of pernicious anæmia, the condition was extremely rare in childhood, and the absence of reticulocytosis and the presence of free hydrochloric acid in the gastric juice rendered this diagnosis unlikely. It was not an aplastic anæmia, in which no regeneration would have taken place at all. Despite the absence of a family history, he thought that the diagnosis of acholuric jaundice must be considered, because while the usual blood picture was that of a microcytic anæmia, icterus, urobilinuria and even a pernicious type of blood picture could occur. He thought, however, that the history of recurrent tonsillitis for a year was most important, and he presented the case as one of hæmolytic macrocytic anæmia due to chronic sepsis.

Obituary.

WILLIAM DAVID KERR MACGILLIVRAY.

Dr. William David Kerr Macgillivray, whose death was recorded recently in this journal, was a man of sterling character. His death has been a blow to the people of western New South Wales, whom he served for so many years. He was best known beyond the district of Broken Hill as an ornithologist, careful in observation and tireless in his searches and explorations.

It is not intended to recount the facts of his early career; this has been done by Dr. Mackay, whose appreciation is published below. A tribute, however, must be paid to the character and worth of the man. He was a clinician with insight and knowledge; he was invariably kind, and he won the confidence of all with whom he came into contact. Nowhere was this more noticeable than in France, where he was attached to the Second Australian General Hospital. For a space also he was anæsthetist to a surgical team. The most grateful recollections centre round association with him; his quiet assurance inspired confidence in his colleagues and his patients; and he was the consoler of many with troubled spirits and anxious minds.

Dr. J. G. Mackay writes:

William David Kerr Macgillivray was born at Kallara Station, River Darling, on November 27, 1867.

In 1870 the family removed to Eddington Station on Eastern Creek, in the Gulf country. He came to Melbourne in 1877 and was educated at St. Kilda Scotch College. He matriculated in 1885 and began his medical course at the Melbourne University in 1886. At the completion of his course he was resident at the Melbourne and Children's Hospitals. He practised in Coleraine, Victoria, in 1896, remained there for two and a half years, then removed to Hamilton and came to Broken Hill in 1901.

He left Australia in 1916 on active service, was stationed at Sutton Veny clearing hospital, and later on in France at Number 2 Australian General Hospital, Wimmereaux, and at Number 3 Casualty Clearing Station, Doullens. He returned to Australia after the armistice with the rank of major.

He was a man of many parts. In his professional work he was a clever and able surgeon and a sound physician. His industry was indefatigable and his energy boundless.

His personal charm and equable temperament endeared him not only to his large *clientèle* of patients, but also to all with whom he came in contact. When at school he won the prize for the most popular boy and the same attributes remained with him through his life.

Outside his professional work his hobby was ornithology, and I do not know of any name in Australia which could command more respect on that subject this his. His ideal holiday was to load up a motor truck with food and equipment and then vanish into the desert or unknown parts of south-west New South Wales, Queensland, and the adjoining parts of South Australian country in search of rare birds, insects and animals. He also made several trips to the Barrier Reef at various times to extend his knowledge of natural history.

I had been very closely associated with him from our school days until a few years ago, and can only join in the grief which I know will be felt in the "West of the Darling" at the passing of such a splendid character and "white" man.

He leaves a widow, son and daughter to mourn his loss.

Mr. Tom Iredale, of the Australian Museum, Sydney, writes:

In the person of W. D. K. Macgillivray the foremost field ornithologist in Australia passed away. To a name shining bright among the annals of British bird study and in the early history of Australian scientific investigation the Australian relative added lustre. One hundred years ago the Scotch W. Macgillivray made that name famous, and his son, as a youth, accompanied Forbes on the *Fly* into Australian waters. Later, that son, still very young, revisited Australia as chief naturalist on the *Rattlesnake*. No better naturalists have ever lived, and our recent friend, W. D. K. Macgillivray, though not a direct descendant, was closely related, and merits as high praise. Of Scotch ancestry, he brought to bear upon the study of ornithology, his favourite group in zoology, all the patience, caution, and perseverance of that race. Thus all his work, as that of his namesake, is absolutely reliable, and it will always be a matter for regret that Macgillivray published so little of his encyclopædic knowledge. Every word he wrote is of extreme value, because he would not write anything down until he had absolutely satisfied himself; and of his own work he was the keenest of critics.

It is a trite saying that naturalists are born, not made; and in this case it was absolutely true. When he was scarcely more than an infant Macgillivray was developing nature study on the station in the Gulf country where his parents lived. Through the disparity of age between himself and his elder brothers, aboriginal children became his playmates. These revealed to him the secrets of Nature, but unlike them, he collected the butterflies, beetles and other objects of interest, and actually formed a little museum of his own. At the tender age of ten he had to come to more civilized places for schooling, and he brought his treasures with him, but he did not know how to pack, and when Townsville was reached they were in such a condition that he was persuaded to discard them. This, however, did not discourage the young naturalist, but spurred him on, and he was always improving his museum. Throughout his long, very busy and successful life he continued as a collector, and a very fine series of natural history objects was brought together.

Although his name is best known to zoologists through his work in ornithology, he was interested in everything in nature, zoological, botanical and geological. Indeed it is possible that he was as good a botanist as he was a zoologist, and that is saying a great deal. While not dour, he was reserved and somewhat slow of speech, a better listener than talker, although often he knew more than his informant. His school and college days were spent in Victoria, where he met the Victorian naturalists, and he later practised in that State, meeting Dr. D'Ombrain,

now of Sydney. These became fast friends, D'Ombrain being also a devotee at the shrine of ornithology. In 1901 Macgillivray settled in Broken Hill, New South Wales, and he spent the rest of his life there. From that centre he made trips in every direction, learning everything about the natural history of the district. Then, with the advent of motor cars, longer trips were made right up into his parental Gulf country, where his brothers still lived. The meeting of the Australian Ornithologists' Union at Brisbane made a great change in his ornithological studies. He became enamoured of the beauty of the Great Barrier Reef and travelled along it, studying its most interesting bird life. Again and again he visited it and projected a full account of its birds, upon which he was engaged right up to his death. It is to be hoped that he has left his notes in a manner suitable for publication, as his knowledge would be of inestimable value to all of us.

The most notable achievement of Macgillivray was made with the assistance of MacLennan, whom he commissioned to explore northern Queensland. A report of mysterious birds in the Pascoe River district was investigated and two large parrots, quite new, were found, the most remarkable addition to the birds of Australia in the past fifty years. MacLennan was a personal friend whom he had taken with him on his local trips round Broken Hill.

Americans recognized his ability in electing him a Corresponding Fellow of the American Ornithologists' Union, while in Australia he was selected as President of the Royal Australasian Ornithologists' Union, the highest honour possible; other societies also honoured him.

Everyone who met him realized his sincere love of nature, his solid workmanship, and he will long be remembered through his careful papers, which are worthy of emulation and deserving of the highest praise.

Correspondence.

PATHOLOGICAL EXAMINATION OF TUMOURS.

Sir: We were interested to read Dr. H. G. R. Darling's letter in your issue of June 17 and regret having delayed so long in making any reply. Dr. Darling desires to sound a word of warning with regard to the limitations of biopsy study in the diagnosis of cancer. He draws attention to Circular 716 of the Ministry of Health, in which the diagnosis of cancer of the breast is considered and in which the committee says that the removal of portions of tissue for microscopic examination with subsequent radical removal of the breast, where necessary, involves grave risk of dissemination of cancer cells.

In our communication to the Fourth Australian Cancer Conference, which was subsequently published in your issue of May 20, we did claim that in our opinion biopsy material should be removed wherever possible and that this could be done without prejudice to the patient. It has been the custom for many surgeons in Adelaide to remove material for biopsy study, and a considerable mass of experience has been gained over a period of years. Our opinion has been based on this experience and not by following tradition or on any preconceived hypothesis. In the surgical treatment of cancer, material becomes available for histological study, but with the advent of treatment by irradiation diagnosis cannot be controlled without the removal of tissues for biopsy study. Experience should, wherever possible, be based on controlled diagnosis.

Dr. Darling draws attention in his letter to the fact that the dissemination of cancer cells differs in various regions of the body. At the same time his main argument against biopsy study is based on the opinion of a committee reporting on cancer of the breast only. It may be remembered that in our communication we said: "The fact that the tissue is removed for biopsy study presupposes that treatment will be proceeded with as soon as possible. In actual practice at the Adelaide Hospital the biopsy material is usually removed at the time of treatment." The treatment of cancer is at all times a matter of urgency, and the histological examination of biopsy material by us is carried out either at the time of operation, within an hour or so, or within twenty-four hours of the removal of the tissue, according to circumstances. The employment of histological methods involving delay of the best part of a week is not justifiable when treatment depends on the result of the examination.

The examination of breast tissues does involve special consideration. It is the practice of many surgeons, when any doubt as to diagnosis exists, to excise the whole tumour and make a careful macroscopic examination before proceeding. When treatment is to be by irradiation, this practice is not followed. The tumour is frequently not

readily accessible for biopsy study and, therefore, these circumstances were not given special consideration in our communication. We do believe, however, that the diagnosis of breast tumours should be controlled by histological examination when treatment is to be by irradiation. We believe that there may be distinct advantages to be gained by local removal of the tumour by surgical means before or soon after irradiation has commenced. There would appear to be little advantage in relying entirely on the action of the rays to destroy all the cancer cells in a well defined mass in the breast. If the mass is removed surgically, there is less risk of some of the cells remaining undestroyed, while the main action of the irradiation would be directed against the cells permeating the surrounding tissues, with more prospect of success.

It is the practice of the gynæcologist to remove with the curette from the uterine cavity, or to cut from the cervix, tissues for biopsy study. We are not aware that this practice has been regarded as involving definite risk of disseminating cancer cells. At the Institut du Radium de Paris biopsy is carried out as a routine on every patient. The five-year cure rate of patients with carcinoma of the cervix treated in this institution for the years 1923 to 1926 is for Stage I 86%, and for Stage II 42%. There would appear to be no evidence that removal of tissue for biopsy in these cases has increased the difficulty of treatment or increased the risk of dissemination of cancer cells.

We do admit that there are times when the removal of tissue from accessible neoplasms for biopsy study scarcely seems to be justifiable. Most of us believe, mainly through current teaching, that surgical interference with a melanotic tumour is a dangerous proceeding. It may be of interest to some of your readers to know that tissues for biopsy study have been removed in quite a number of cases of malignant melanoma of the skin in patients coming to the cancer clinic here and that at no time has this surgical interference produced any untoward result. We are pleased to be able to state that of eighteen cases of malignant melanoma treated in this clinic, three are alive and free from symptoms from two to three and a half years after the commencement of treatment, in spite of biopsy material having been removed. In sixteen of these eighteen patients dissemination had already occurred before treatment was started.

B. T. Simpson (*Canadian Medical Association Journal*, January, 1933, page 62) discusses the problem of biopsy in cancer diagnosis. The original paper is not accessible here, but according to an abstract in *The British Medical Journal*, May 6, 1933, page 78, he is reported as saying that clinical observations and laboratory experiments go to prove that cutting a malignant tumour does not stimulate metastasis. He states, further, that as regards bone lesions, the procedure is still open to controversy and that he does not employ it for these or for melanomata.

As treatment becomes more efficient the objection to biopsy study, which appears to be mainly theoretical, will probably disappear. It is important, however, that efficient treatment should be instituted at the earliest possible moment, and if the treatment is by irradiation, this can be started before or at about the same time as the removal of the tissue for biopsy study.

Dr. Darling is not correct in assuming that our opinions were sponsored by the Fourth Australian Cancer Conference. No definite opinion was expressed by the conference. We have to take full responsibility for our opinions, but they are supported by, and to a large extent result from, the experience of the Cancer Clinic of the Adelaide Hospital.

Yours, etc.,

Lionel B. Bull.
B. S. Hanson.

Laboratory and Radium Department,
Adelaide Hospital,
September 16, 1933.

THE PROBLEM OF GASTRIC CARCINOMA.

Sir: In a scientific discussion Dr. Rutherford Darling should confine himself to the issue at point and realize that abuse is not criticism.

In dealing with the detection of occult blood we are concerned here only with the benzidine test and the Gregersen's slide (benzidine) test, though one must remember that various adjuvant tests may be necessary.

It is important not to be confused by the term occult, the test in its essential is the detection of an iron-containing blood pigment.

Dr. Rutherford Darling desires proof through scientific and logical methods that the conclusion of Dr. A. G. Ogilvie:[1] "A blue or blue-green colour with Gregersen's test obtained before thirty seconds is proof positive of the presence of occult blood in the stools irrespective of the patient's diet" is faulty and erroneous.

Dr. Ogilvie makes the following statements in the course of his experiments.

First series of experiments: Roast beef and pickled herring gave immediate positive reactions, while mince gave a 2+ reaction, that is, a definite pale blue colour in twelve to fifteen seconds.

Second series of experiments—experiments on a normal individual:

(*a*) A faint positive test was given by Gregersen's method on a diet including roast meat.

(*b*) A diet on one day, containing pickled herring, gave a negative result.

(*c*) Three ounces of raw meat failed to give a positive reaction.

(*d*) Two ounces of cooked ox blood gave no reaction, but if uncooked was just sufficient to give a definite reaction.

Third series of experiments—experiments on thirty pathological cases. Fourteen negative and sixteen positive results were recorded; bleeding on brushing the teeth occurred in practically every case. Of the negative cases, seven were on a full diet; of the positive cases seven gave a faint coloration after thirty seconds (1+ reaction); the rest gave more definitely positive results.

"Of the seven patients who gave a faint 1+ reaction, in five the diet could not be excluded as a possible cause, but in the two remaining, a case of chorea and a case of acute rheumatic fever, the bleeding could be fairly attributed to the alimentary canal." It may be noted that of these positive patients some at least had no intestinal lesion.

Other experiments: Two patients with pernicious anæmia who had been taking iron continuously for over three months, gave consistently negative results. A patient with pernicious anæmia given bone marrow gave a 3+ result, even though no intestinal lesion was found at necropsy. This, he states, seems to illustrate a definite fallacy.

Ogilvie summarizes his experiments and comes to the conclusion that a positive Gregersen test obtained within thirty seconds is proof positive of the presence of occult blood in the fæces, irrespective of the diet. Ogilvie himself quotes four cases where a positive test was due solely to diet (compare above).

From Ogilvie's experiments we find that a positive Gregersen test is sometimes given by occult blood, sometimes not given by occult blood, sometimes given by certain substances in the diet, and sometimes not given by the same substances in the diet.

Ox blood contains approximately the same amount of hæmoglobin as human blood (cow's blood, 12·6 grammes *per centum* of hæmoglobin). If less than two ounces of ox blood is taken, a positive Gregersen's test will not be given by the time the blood is passed in the fæces. This surely should be interpreted as not meaning that Gregersen's test is independent of the diet, but that it is insensitive to large amounts of reacting substances, that is, a positive Gregersen's test depends on the presence of a high concentration of blood in the fæces. Clinically this may mean many a missed early diagnosis.

Harrison's[2] conclusions as to the relative value of the tests is as follows, referring to the benzidine test as Method 1 and Gregersen's slide (benzidine) test as Method 2.

"Method 2 really amounts to making the technique less sensitive so as to avoid the preparation of the patient essential in Method 1. In common with others, the writer has sometimes found the slide method negative when the test tube method has given definitely positive results which have been proved to be significant. His conclusion is that the first method is the method of choice, but that the second is a useful bedside test of more limited value and which should be employed only when the proper laboratory facilities for Method 1 are not available."

So much for the scientific aspect of Dr. Ogilvie's results. Dr. Rutherford Darling's appeal to logic, if possible, is more unfortunate.

Mallone[3] states "Mill's Method of Single Difference", that is, the method of experiment, thus:

"When the addition of an agent is followed by the appearance, or its subtraction, by the disappearance of a certain event, other circumstances remaining the same, that agent is causally connected with the event."

This canon refutes Dr. Ogilvie's hypothesis.

Yours, etc.,

"Pathologist."

September 18, 1933.

References.

(1) A. G. Ogilvie: *The British Medical Journal*, April 23, 1927, page 755.
(2) G. A. Harrison: "Chemical Methods in Clinical Medicine", 1930.
(3) Mallone: "Introductory Text Book of Logic."

DIAGNOSTIC SWABBINGS IN DIPHTHERIA.

Sir: In The Medical Journal of Australia of September 9, page 358, in the discussion of a paper by Dr. Leslie Hurley, read before the Victorian Branch, Dr. Derham is reported as supporting Dr. Hurley in "referring to the taking of diagnostic throat swabs in cases of diphtheria as being useless or worse than useless from the therapeutic point of view".

Incidentally I cannot find any reference to diphtheria or throat swabs in Dr. Hurley's paper which is published in the same number of the journal. Perhaps in revising the paper for publication he cut this out as being too sweeping a generalization, or likely to mislead the casual reader.

If this was indeed his second thought, I am in accord with him. It is true that the diagnostic swabbing is of little value as a guide to treatment. Treatment should be begun at once on the clinical evidence. Every hour of delay in giving antitoxin, for example, while waiting for the bacteriological report, may be prejudicial to the patient.

But for confirmation of the clinical diagnosis swabbings are still essential. Such confirmation is not merely of academic value. On it may depend serious consequences in regard to isolation, school attendance, occupational restrictions (for example, in a milk vendor and dairyman).

It is true that the swabbing results are sometimes surprising. An obviously diphtheritic throat may give a "negative" and an innocent looking surface a positive result. Some of the "positives" may be due to diphtheroids and some of the "negative" anomalies to defective making of the swabbing. The swab has not been rubbed on the actual diphtheritic surface or the material collected on the swab has not been transferred to the culture medium.

Even with these limitations diagnostic swabbings cannot be dispensed with. And probably Dr. Hurley also is of this opinion.

Yours, etc.,

R. Mullard,
Clinical Tutor in Infectious Diseases, University of Sydney.

135, Macquarie Street,
Sydney,
September 20, 1933.

THE USE OF IODINE IN THYREOTOXICOSIS.

Sir: I have read, with pleasure and profit, Dr. Poate's article on the "Use of Iodine in Thyreotoxicosis". It is so clear and practical that I hesitate to criticize anything in it. However, I wish that the author had emphasized certain points in the paper, and I venture to refer to them here in this letter.

All through his article he clearly and most necessarily points out the uses of iodine, and its limitations, and says, *à propos* of the treatment of primary thyreotoxic conditions that: "When the iodine deficiency is made up in these patients, the clinical results in most cases are very striking, for the time being." Quite so, but I wish that the last four words had been in italics, for the qualification is vital. Similar prominence, when he is speaking of secondary thyreotoxicosis, might also have been given to the statement that: "Whenever iodine is given, the consistency of the thyreoid gland must be watched carefully, as, once it becomes firm, the dose should be reduced to a minimum or discontinued." On page 413 the two paragraphs dealing with the moment when the patient is found to be fit for operation, after the very important preliminary treatment, and describing what evils may follow unwise persistence in the use of iodine, might have been given greater prominence. Iodine, in the cases Dr. Poate is dealing with, has its great value for a certain time; continued after the happy moment, harm follows. Somewhat, indeed, it reminds me, in a not very analogous way, of the use of lactate of lime in chilblains. If that drug has not done what one wishes in eight days or less, it should be left off. Dr. Poate rightly advises the use of Lugol's solution, and no doubt it is the best form we have just now in which to administer iodine. Why? Because it has iodide of potash as a constituent. Dr. Poate elsewhere refers to the need of the alkali, but it would have been quite proper to have accentuated the inherent virtue of Lugol's solution because of its constituent iodide of potash. I remain not wholly convinced that belladonna is an evil drug, but Dr. Poate, I believe, has good practical reasons for condemning it. As to sedatives, I am afraid that I remain unconvinced of the virtues of "Barbenyl", "Luminal" *et cetera*, nor do I quite accept his praise of paraldehyde by the mouth. Lastly, I wish very much than Dr. Poate had told us clearly his position with regard to the anæsthetic to be used when an operation on the gland is called for. Rightly he has pointed out the evils of an inhalation anæsthesia which induces cyanosis, but in his section on treatment I am sure we would all have wished to hear whether he uses local or general anæsthesia. If the latter, in what form? Perhaps he might add to his excellent article some amplification of it on the lines I have indicated.

Yours, etc.,

R. Scot Skirving.

Sydney,
September 22, 1933.

Sir: H. R. G. Poate, in his article in your issue of September 23, speaks of "a deficiency in the iodine content of thyroxine" (page 413).

This, of course, can never be. Thyroxine is a definite chemical compound, $C_{15}H_{11}O_4I_4N$.

Yours, etc.,

G. A. Sampson, M.B., Ch.M.

Brisbane,
September 25, 1933.

INFLUENZA.

Sir: I wonder if any other of your readers has noticed the following symptom in influenza patients during the present epidemic.

When the patient has been undisturbed for some time, there is slight or sometimes well marked dulness over a lung area. On placing the bell of the stethoscope over the dull spot, while the patient takes a deep inspiration a crepitation is heard. The sound resembles that produced

by stripping gummed fabric from a resonating surface, the gum being "tacky". It occurs only during one or two respirations following the rest period. When the crepitus is no longer heard the dulness will be found to have disappeared.

Long after the temperature has become normal and the patient is progressing towards recovery the symptom may persist.

The character of the sound in association with the transient dulness leads me to infer that it is of vesicular origin.

The effect is probably produced by a gluing together of vesicular walls. It indicates that even during the acute stages one should encourage deep breathing and, by varying the position in which the patient lies, prevent any part of the chest from being held immobile during long periods. At regular intervals he should be induced to undertake over-breathing exercises. In convalescence, the sooner he returns to active life the better.

Possibly the gluing in the vesicles may account for the proneness to relapse in those patients, and some cases of free hæmorrhage may well be produced by tearing the glued walls apart.

Yours, etc.,

William P. Kelly.

38, MacGregor Terrace,
Paddington,
Brisbane,
Undated.

Diary for the Month.

Oct. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Oct. 13.—Queensland Branch, B.M.A.: Council.
Oct. 17.—New South Wales Branch, B.M.A.: Ethics Committee.
Oct. 18.—Western Australian Branch, B.M.A.: Branch.
Oct. 18.—Victorian Branch, B.M.A.: Clinical Meeting.
Oct. 19.—New South Wales Branch, B.M.A.: Clinical Meeting.
Oct. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Oct. 25.—Victorian Branch, B.M.A.: Council.
Oct. 26.—South Australian Branch, B.M.A.: Branch.
Oct. 26.—New South Wales Branch, B.M.A.: Branch.
Oct. 27.—Queensland Branch, B.M.A.: Council.
Nov. 1.—Western Australian Branch, B.M.A.: Council.
Nov. 2.—South Australian Branch, B.M.A.: Council.
Nov. 3.—Queensland Branch, B.M.A.: Branch.
Nov. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.

Medical Appointments.

Dr. A. R. Southwood (B.M.A.) and Dr. E. A. Johnson have been appointed members of the Advisory Committee under the *Food and Drugs Act*, 1908, South Australia.

* * *

Dr. H. McI. Birch (B.M.A.) has been appointed Deputy Superintendent of the Parkside Mental Hospital, and also Deputy Superintendent of the Hospital for Criminal Mental Defectives, under the provisions of *The Mental Defectives Act*, 1913, South Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

Children's Hospital (Incorporated), Perth, Western Australia: Junior Resident Medical Officers.

Hobart Public Hospital, Hobart, Tasmania: Junior Resident Medical Officers.

Metropolitan Infectious Diseases Hospital Board, Adelaide, South Australia: House Physician.

New South Wales (Community) Hospital, Incorporated, Sydney, New South Wales: Physician for Children's Clinic.

Parramatta District Hospital, Parramatta, New South Wales: Junior Resident Medical Officer.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
South Australian: Secretary, 267, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
New Zealand (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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